THE POTENTIAL ROLE OF WILDLIFE IN THE SPREAD AND CONTROL OF FOOT AND MOUTH DISEASE IN AN EXTENSIVE LIVESTOCK MANAGEMENT SYSTEM

A Dissertation

by

LINDA HIGHFIELD

Submitted to the Office of Graduate Studies of
Texas A&M University
in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

August 2008

Major Subject: Biomedical Sciences

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Approved by:

Chair of Committee,	Michael Ward
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ABSTRACT

The Potential Role of Wildlife in the Spread and Control of Foot and Mouth Disease in an Extensive Livestock Management System (August 2008)

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Chair of Advisory Committee: Dr. Michael Ward

Foot and mouth disease (FMD) is a highly contagious viral infection that affects all Artiodactyls (cloven-hoofed) species. The United States has been free of FMD since 1929, and the entire population of cloven-hoofed species is therefore susceptible to FMD virus infection. In the face of an outbreak, it is crucial that appropriate control measures be applied rapidly to control the disease. However, in most cases decisions on mitigation strategies must be made with little current or empirical data and in the context of political, economic and social pressures. Disease spread models can be used to evaluate the design of optimal control strategies, for policy formulation, for gap analysis and to develop and refine research agendas when disease is not present. This research project is designed to *investigate the potential role of wildlife (deer) in the transmission and spread of FMD* in an extensive livestock management system in southern Texas. The spread of FMD was simulated in white tailed deer populations using a Geographic Automata model. Past research has focused primarily on modeling the spread of FMD in livestock populations. There has been limited research into the potential role of wildlife in the spread and maintenance of FMD, specifically in the United States and using a

spatial modeling approach. The study area is a nine-county area located in southern Texas, bordering Mexico. It is a region of concern for the introduction of foreign animal diseases, particularly through the movement of wild and feral animal species. It is both a strategic location and is generally representative of the many similar eco-climatic regions throughout the world. It is an ideal model landscape to simulate FMD incursions. In this research project, the potential spread of FMD is simulated based on various spatial estimates of white tailed deer distribution, various estimates of critical model parameters (such as the latent and infectious periods), seasonal population variability and in the face of potential pre-emptive mitigation strategies. Significant differences in the predicted spread were found for each group of simulations. The decision-support system developed in the studies described in this dissertation provide decision-makers and those designing and implementing disease response and control policy with information on the potential spread of a foreign animal disease incursion with a likely wildlife reservoir. Use of such a decision-support system would enhance the disease incursion preparedness and response capacity of the United States.

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CHAPTER I

INTRODUCTION

Foot and mouth disease (FMD) is a highly contagious viral infection that affects all Artiodactyla (cloven-hoofed) species, including cattle, deer and pigs. FMD is considered one of the most serious livestock diseases and is found in two-thirds of the Office International des Epizootes (OIE) member countries [74]. The FMD virus is an apthovirus within the family picornaviridae. There are seven known serotypes (A, O, C, South African Territories (SAT) 1, 2, and 3 and Asia 1 [61]), and there is no cross protection across serotypes [74]. Serotypes A, O, and C have been identified in Europe and South America. FMD is endemic in most of Africa and Asia and parts of South America. FMD is not endemic in Europe, Australia, New Zealand or North America; however sporadic outbreaks in Europe have occurred, mostly as a result of spread from the Middle East [74]. The United States has been free of FMD since 1929, and the entire population of cloven-hoofed animals (cattle, sheep, goats, swine and wildlife) is therefore susceptible to FMD virus infection.

Replication of FMD virus is extremely rapid in the upper respiratory tract or lung. The resulting viraemia seeds the infection into the epithelium where secondary replication

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This dissertation follows the style of Veterinary Research.

results in vesicle formation and shedding of the virus [40, 81]. The incubation period may be as short as 2−3 days or as long as 14 days [32]. Infected animals may become infectious before exhibiting clinical signs [12]. The virus is excreted as a particulate for several days before the animal begins to recover [16]. The amount of virus excreted varies by species, with pigs excreting substantially larger amounts than cattle or sheep [80]. Pigs are also highly susceptible to oral infection and as such, can be classified as amplifier hosts (high susceptibility and high infectiousness). Cattle are considered indicator hosts because they have high susceptibility but lower infectiousness. Sheep are considered maintenance hosts because they often exhibit mild or inapparent clinical signs [2]. The virus may be transmitted via direct (movement, aerosols) and indirect (fomites and people) contact with infected animals.

FMD virus has been observed to survive for long periods of time in favorable environmental conditions (neutral pH (~7), low temperatures (4^{o}C), presence of organic matter). Environmental exposure (shared habitat) may be a concern for infection of susceptible livestock in certain areas. At 4^{o}Celsius with neutral pH, it has been observed to take 18 weeks for 90% virus inactivation [73]. As an aerosol, FMD survives best when the relative humidity is above 70%; it will not survive below 55% relative humidity [73]. Following slaughter, FMD virus is inactivated within 48 hours provided the pH drops below 6.0 [16]. Freezing tissues containing the virus may ensure its survival for a period of years. The virus may also survive longer in lymph nodes and bone marrow [16].

FMD virus infection is characterized by vesicular lesions on the hooves (intradermal pad and coronary band) and mucosa of the mouth (tongue and soft palate). Vesicles typically contain clear or straw-colored liquid prior to rupture [16]. The lesions are readily identifiable in cattle and swine, but almost indistinguishable in sheep. The disease severity ranges from mild clinical signs or illness to death, in areas where the disease is not endemic. In endemic regions, the infection may be mild. Mortality in adult cattle is low (generally <5%), however it is much higher in younger cattle (up to 90%) [2]. After recovery, approximately 50% of cattle become carriers [73]. The duration of the carrier state varies by species: >3 years in cattle, 9 months in sheep, and four months in goats [2]. The role of carrier animals in the spread of FMD is unclear [73]. There is speculation that carrier animals have played a role in outbreaks [67, 9], however, no spread of disease from carriers to susceptible animals has been observed under controlled conditions [5, 67].

The United States has a number of susceptible wildlife species, including white tailed deer, mule deer, antelope, elk and feral pigs. The severity of the disease in *Cervidae* (deer) varies from inapparent or mild in some species to more severe in others [74]. Deer have been infected both naturally and experimentally [64, 31, 35, 74], and deer-to-deer, deer-to-cattle and cattle-to-deer transmission has been observed [82]. Experimentally infected white tailed deer exhibited intermediate disease severity compared with susceptible livestock species (such as cattle, sheep and goats) [64]. Several species of

deer are among the most commonly FMD-infected wildlife under field conditions, and deer are believed to play an important role in the epizootology of the disease [74].

The United States has been free of FMD since 1929, following a number of outbreaks in California and Texas during the 1920's. In 1924, an outbreak in California started in pigs (swill fed), spread to grazing cattle and subsequently infected deer across the central portion of the state. It required 2 years to eradicate the disease from the local deer population, and more than 22,000 deer were slaughtered [47, 64]. Approximately 10% of the slaughtered deer during the 1924 outbreak displayed typical signs of FMD infection [64].

FMD infection in wildlife has also been a concern in more recent FMD outbreaks. During the 2001 FMD outbreak in the U.K., it was feared that a number of the deer species in the country (red, roe, fallow) might become infected and potentially act as a reservoir for the disease [16, 85]. A similar concern was also expressed in the Netherlands during the 2001 FMD outbreak [27, 85]. However, evidence of infection in wild deer was not observed in either of these recent outbreaks, although there were reports of wildlife displaying signs of infection [27]. Extensive serosurveillance was conducted after the outbreak, but deer were not tested as part of the surveillance program [27]. Due to the nature of the cattle industry in Europe, a lack of contact between deer and livestock in these countries may have averted a disastrous situation from occurring [27].

There are numerous areas of the United States where livestock are extensively grazed and the potential for interaction with susceptible wildlife species, such as white tailed deer, is high. Deer move through and forage in fields between farms and enter premises with animal feed and slurry [85]. In addition, supplemental feeding of white tailed deer for hunting purposes is a common practice in many areas of the U.S. [6]. Deer densities in parts of Texas are very high, and most deer inhabit private land [56]. As the result of extensive land use change, deer populations in Texas have formed metapopulations with high deer densities, increased contact between deer populations and potentially the risk of disease transmission to domestic livestock [56].

Epidemic models represent an important tool to aid decision-making and epidemic response to foreign animal disease incursions. In the face of an outbreak, it is crucial that appropriate control measures be applied rapidly to control the disease. However, in most cases decisions regarding mitigation strategies must be made with little current or empirical data and in the context of political, economic and social pressures. Disease spread models can give guidance on the probable extent and time span of an outbreak. They can also be used to evaluate the design of optimal control strategies, for policy formulation, for gap analysis and to develop and refine research agendas when disease is not present.

The conditions under which wild and feral animal species might become reservoirs of FMD virus, following an incursion into a country free of disease, are unknown. However, several factors – including population density and distribution, habitat requirements, social organization, age structure, home range, and barriers to dispersal – are likely to be important. The most recent outbreaks of FMD in the United States, which involved wild (deer) and feral (pigs) animal species, occurred in the 1920s. Because of the paucity of information and experience with FMD in reservoir species, simulation modeling is perhaps the only option for exploring the impact of an FMD virus incursion and therefore developing response plans and formulating policy [86].

Although spatial models using differential equations have been developed to describe the farm-to-farm spread of FMD virus [34, 49, 68, 7], these models have generally ignored the involvement of feral and wild animal species. Models of FMD virus spread that do involve these potential host species [19, 72] have not included the spatial component of spread. Artificial life models, such as Geographic Automata that explicitly incorporate spatial relationships, are an alternative modeling approach [21]. These models of physical systems treat space and time as discrete units and interactions occur between local neighbors [91]. Geographic Automata are generalizations of cellular automata that are not restricted to a regular lattice of cells (geographic locations) and can model complex spatial interactions. Each population interacts with neighboring populations based on a set of rules and states at earlier time steps. The repetitive application of transmission rules within this local neighborhood allows the replication of complex

spatial behavior such as occurs in disease outbreaks. Geographic Automata models can deal with complex initial conditions and geographical boundaries, are relatively simple to construct and understand, are more general than differential equations, do not depend on generalized probability distributions derived from observations or hypothetical data, and are computationally efficient.

Following detection of an incursion of FMD virus in a country previously free of disease, the application of appropriate control measures is a decision that needs to be made rapidly yet with little current or empirical data. In addition, political, economic and property rights issues may also guide policy decisions regardless of what is deemed to be the most effective strategy to reduce the spread of FMD. Information from model outputs that provides guidance to the probable extent of an outbreak and its time span are invaluable for decision-makers implementing disease control measures in the face of external pressures. Nonetheless, such models need to be developed, validated and tested prior to emergency situations. Strong links between disease modelers, policy and decision-makers also need to exist *a priori*. Models can serve not only as response and decision-making tools but also as avenues to increase awareness and collaboration with stakeholders.

This research project is designed to *investigate the potential role of wildlife (deer) in the transmission and spread of FMD*. The spread of FMD will be simulated in white tailed deer populations using a Geographic Automata ("SIRCA") model. Past research has

focused primarily on modeling the spread of FMD in livestock populations. There has been limited research into the potential role of wildlife in the spread and maintenance of FMD, specifically in the United States and using a spatial modeling approach. The study area selected for this research project is a 9-county area located in southern Texas, bordering Mexico. It is a region of concern for the introduction of foreign animal diseases, particularly through the movement of wild and feral animal species. It is both a strategic location and is generally representative of the many similar eco-climatic regions throughout the world. It is an ideal model landscape to simulate FMD incursions. This study will combine the expertise of veterinarians, epidemiologists, geographers and pathobiologists through a collaborative partnership with the Foreign Animal and Zoonotic Disease Defense Center, generating information on the potential role of deer in the spread and maintenance of FMD and steps that might be taken to reduce this impact.

CHAPTER II

REPRESENTATION OF ANIMAL DISTRIBUTIONS IN SPACE: HOW GEOSTATISTICAL ESTIMATES IMPACT SIMULATION MODELING OF FOOT AND MOUTH DISEASE SPREAD

Modeling potential disease spread in wildlife populations is an important tool for predicting, responding to and recovering from a foreign animal disease incursion. To make spatial epidemic predictions, the target animal species of interest must be represented in space prior to modeling disease spread. A series of simulation experiments were conducted to determine how estimates of the spatial distribution of white tailed deer impact the predicted magnitude and distribution of foot and mouth disease outbreaks. Outbreaks were simulated using a susceptible-infected-recovered geographic automata ("Sirca"). The study region was a 9-county area of southern Texas. Methods used for estimating deer distributions included dasymetric mapping, kriging and remotely sensed image analysis. The magnitudes and distributions of predicted outbreaks were evaluated by the median number of deer infected and median area affected (km^2), respectively. The estimation methods were further evaluated for similar predictive power by comparing model predicted outputs with Unweighted Pair Group Method with Arithmetic Mean (UPGMA) clustering. There were significant differences in the estimated number of deer in the study region, based on the estimation procedure

used (range: 385,939- 768,493). There were also substantial differences in the predicted magnitude of the foot and mouth disease (FMD) outbreaks (range: 1,563–8,896) and the number of locations affected (range: 56–447 km^2) for the different estimated animal distributions. UPGMA clustering predicted two main groups of distributions, and one outlier. We recommend that one distribution from each of these two cluster be used to model the range of possible outbreaks. Methods included in the first cluster (such as county-level disaggregation) could be used in conjunction with any of the methods in the second cluster, which included kriging, NDVI split by ecoregion, or disaggregation at the regional level, to represent the variability in the model predicted outbreak distributions. How animal populations are represented in space needs to be considered in all spatial disease spread models.

1. Introduction

The aim of this research was to evaluate the sensitivity of epidemic model predictions to estimated spatial distributions of wildlife species. This research provided critical insight into the impact that estimated spatial distributions have on modeling predictions. This is important because modeling predictions may be used to guide policy and evaluate mitigation strategies prior to an outbreak [86, 96]. Modeling may also be used during an outbreak to inform response strategies. There are many questions that may be asked by policy- and decision-makers either before or in the face of an outbreak [86]. Two possible questions ask the predicted average outbreak size and the “best” or “worst” case

scenario [86]. The former question relates to the mean or median predicted outbreak size, which is useful for investment decisions and ranking the importance of foreign animal disease outbreaks. The latter question addresses the minimum and maximum predicted outbreaks, information that can be used for potential resource allocation. Decision- and policy-makers may also be interested in specific values within the predicted distribution of outbreaks [86].

Since epidemic predictions are obtained via Monte Carlo simulations, the entire distribution of predicted outbreaks should be considered when making decisions, rather than focusing on central tendency or variability statistics [86]. Given these considerations, this chapter addresses the impact of 15 different geostatistical methods for estimating the spatial distribution of deer distribution on the predicted outbreak distribution of foot and mouth disease (FMD) in southern Texas.

Foot and mouth disease (FMD) is a highly contagious viral disease of cloven-hoofed animals, affecting all Artiodactyla species, including cattle, deer and feral hogs. It is considered one of the most serious diseases of livestock [74] and the economic devastation caused by FMD outbreaks can be vast, as evidenced by recent outbreaks [99, 45]. The severity of the disease in deer varies from unapparent or mild symptoms in some species to more severe in others [74]. Deer have been infected both naturally and experimentally [64, 31, 35, 74], and deer-to-deer, deer-to-cattle and cattle-to-deer transmission has been observed [35]. Experimentally infected white tailed deer exhibited

intermediate disease severity compared with susceptible livestock species and approximately 10% of those infected in a 1924 outbreak in California displayed signs of FMD infection [64]. Several species of deer are among the most commonly FMD-infected wildlife under field conditions, and they are believed to play an important role in the epizootology of FMD [74].

The United States has been free of FMD since 1929, following a number of outbreaks in California and Texas in the 1920's. In 1924, an outbreak in California started in pigs, spread to grazing cattle and subsequently infected deer across the central portion of the state. It took 2 years to eradicate the disease from the local deer population, with more than 22,000 deer slaughtered [47, 64].

During the 2001 FMD outbreak in the U.K., it was feared that a number of the deer species in the country might become infected and act as a reservoir for the disease [16, 85]. A similar concern was also expressed in the Netherlands during the same time period [27, 85]. However, evidence of infected deer was not observed in either of these more recent outbreaks [27].

In areas of the United States where livestock are extensively grazed, the potential for interaction with susceptible wildlife reservoirs is high [96]. Deer move through and forage in fields between farms, and enter farms with animal feed and waste [85]. Additionally, supplemental feeding of white tailed deer for hunting purposes is common

and may enhance deer-to-deer transmission [96]. Given the widespread distribution of wildlife species susceptible to FMD virus infection and the high potential for interaction with livestock, modeling the spread of the disease in wildlife populations is an important tool in our ability to predict, respond to and recover from foreign animal disease incursions.

To model the spread of FMD in a wildlife population, such as white tailed deer, an estimate of the species spatial distribution is crucial. A variety of spatial estimation methods have been used for modeling the density and distribution of wildlife species, including dasymetric mapping, regression-based approaches, and remotely sensed data.

Dasymetric mapping, also known as surface based demographic data representation, redistributes the population from a aggregate level into either a vector or raster map using ancillary data such as land use or remotely sensed images [65]. Dasymetric mapping provides a means of visually representing a statistical distribution in a Geographic Information System (GIS) using aggregate data [17]. This mapping procedure eliminates the artificial structure of political or arbitrary boundaries that are often placed on aggregate data thereby allowing for representation of a more realistic distribution of the data [17, 54].

Regression approaches that have been used to describe the density and distribution of wildlife species vary widely and include ordinary least squares, logistic, Poisson,

geographically weighted regression and kriging. Kriging predicts values at unsampled locations using the autocorrelation structure in the measured observations and the values of nearby observations, taking into account both distance and geometry [11, 77].

Use of remote sensing in epidemiology is based on the development of a logical sequence linking remotely sensed measures of radiation to measures of a disease and its corresponding vector or host [15]. Perhaps the most commonly used type of remotely sensed data is the Normalized Difference Vegetation Index (NDVI). NDVI data is available from the United States Geological Survey (USGS) Advanced Very High Resolution Radiometer (AVHRR) database. AVHRR data are collected by the National Oceanic and Atmospheric Administration's (NOAA) polar earth-orbiting satellites, which collect data in the visible, near-infrared, and thermal infrared regions of the electromagnetic spectrum. NDVI is associated with photosynthetically active radiation [52].

Epidemics have traditionally been modeled using differential equations (DE) [1, 24, 84]. However, DE models do not directly address the local character of disease spread and cannot handle complex boundary conditions [84]. Geographic Automata (GA) are sequence models, capable of handling non-tessellated data (points). Geographic automata provide an alternative approach to DE based epidemic models. These models treat time and space as discrete and interactions are localized [84]. They have been applied to a wide range of disease spread problems [1, 8, 23, 30, 43, 78, 95].

Susceptible-infected-recovered models are often built into GA to examine the spatial and temporal spread of epidemics [8, 29, 30, 43, 53, 78, 84]. However, this approach has rarely been used to model infectious diseases in wildlife reservoirs. Importantly, the impact of the methods used to derive the spatial reservoirs distribution on epidemiological modeling predictions has, to the authors' knowledge, not been evaluated.

The objectives of this study were: 1) To apply 15 commonly used spatial methods to estimate white tailed deer distributions in southern Texas; 2) Describe the predicted FMD outbreak distribution that might be observed, given the various geostatistical estimation methods used; and 3) Compare the predicted FMD outbreak distributions for each of the geostatistical methods used.

2. Materials and Methods

2.1. Study site

The study site (approximately 24,000 km^2) is an area of southern Texas, bordering Mexico, consisting of nine counties and two Ecoregions (Figures 1 and 2). The Edwards Plateau (EP) in the north and the South Texas Brush (ST) in the south, split the study region approximately in half (Figure 2). Seasonal variation in the overall study region is characterized by hot, dry summers and mild, moist winters.

The EP Ecoregion encompasses the upper half of the study region (Figure 2). The EP is predominately rangeland, characterized by a mix of browse, forbs and grass. The major species include live oak, mesquite, acacia, and prickly pear mixed with areas of grass [88]. The EP is home to the state's largest white tailed deer concentration and the population often exceeds carrying capacity in this area. Population density in this Ecoregion is higher than any other part of the state with an estimated 100 deer per 405 hectares [88]. However, drought is common and it periodically has long term affects on wildlife populations and habitat resources.

The ST Ecoregion comprises the Southernmost region of Central Texas (bordering Mexico) and encompasses the southern half of the study region (Figure 2). The ST Ecoregion is considered a brush community characterized by mesquite, blackbrush, brasil, and other thorny plants [88]. White tailed deer hunting has increased in this region and the vegetation in this area is actively managed to support hunting [88]. Population density for white tailed deer in this Ecoregion is considered moderate with an estimated 29 deer per 405 hectares [88].

2.2. Data source

Data on the number of deer per reporting unit and per county were obtained from the Texas Parks and Wildlife Department (TPWD) [56]. The distribution of deer was estimated using geostatistical methods. All spatial processing was done using ArcGIS

9.0 (ESRI, Redlands, CA) and all surfaces were estimated on a square lattice with a 1 km^2 resolution.

2.2.1. *Disaggregation based on county numbers (DC)*

County-level deer populations were disaggregated based on suitable landuse and estimated carrying capacity within each land use category. Land use categories were derived from the 1992 National Land Cover Dataset (NLCD). The NLCD grid was clipped to the study site and reclassified into suitable categories as follows: forest (land use classes 41, 42 and 43), shrub (classes 50 and 51) and grassland (class 71). The proportion of each land use category in the study area is shown in Table 1. Estimated carrying capacity was derived from expert opinion and yielded values of 0.3 for forest, 0.3 for shrub and 0.1 for grassland for this region. The number of pixels per land use category was multiplied by the carrying capacity as a weighting factor. The number of deer per county was proportionally distributed within land use category based on the weighting factor for each category. The resulting fractional counts of deer at 30 meter resolution were aggregated to a 1 km^2 integer grid.

2.2.2. *Focal smoothing (FS1-FS3)*

To eliminate demarcation lines that appeared in the *DC* method, this data was smoothed using the mean value of cells within a rectangular window around each cell (a focal mean). Three window sizes were used: 10 km^2 (*FS1*), 20 km^2 (*FS2*) and 30 km^2 (*FS3*).

2.2.3. *Disaggregation based on region (DR)*

The number of deer per county was summed to the regional level (9 county total) and disaggregated as described for the *DC* method, but without regard for county boundaries so that demarcation lines that appeared in the *DC* method were eliminated.

2.2.4. *Disaggregation based on reporting unit (DRU)*

Counts of deer per reporting unit were disaggregated using the *DC* method, except the disaggregation was done at the reporting unit scale rather than county scale.

2.2.5. *Kriging and deer redistribution based on land cover (K1-K4)*

The estimated counts for each county were divided by the total area in the county, to derive average densities per km^2. The centroids were calculated for each county and the appropriate density value was joined to the centroid to prepare the data for kriging. An ordinary kriging model (*K1*) was used to create a raster surface of deer density.

The kriged surface was subsequently used to redistribute densities of deer based on land cover type using the NLCD data. The land cover data was reclassified to the following multipliers: shrublands were classified as 1.2, forests were classified as 1.0, and grasslands were classified as 0.8; all other land cover categories (including cropland, residential, and water) were classified as 0. The reclassified land cover raster was multiplied by the kriged surface. This resulted in a raster of deer density that was proportionally distributed within shrubland, forest, and grassland land covers (*K2*).

The redistribution of deer density resulted in areas of higher or lower density than that reported for ecoregions by TPWD. A correction factor was computed for each ecoregion using the ratio of the zonal sum of deer density for each ecoregion obtained from the raster described above to the deer density for each ecoregion (*K3*) reported by TPWD. Finally, to reduce the smoothness of the surface generated by kriging, the estimated deer from kriging were re-distributed based on the proportion of suitable land use within each county (*K4*).

2.2.6. *Disaggregation to farm-boundary (DFB)*

County-level deer population estimates (*DC*) were summed to farm boundaries and a centroid for each farm was used to represent the deer population. Farm boundaries were provided by the United States Department of Agriculture (USDA) Farm Services Agency (FSA) of Texas.

2.2.7. *Linear scaling based on NDVI (LS1-LS4)*

Maximum monthly composite NDVI values for the months of April (*LS1*) and December (*LS2*) 2004 were used to linearly scale deer density in the range 0–30 for each 1 km^2 pixel. April and December were selected because they represent the highest and lowest monthly precipitation, respectively. The estimated densities were summed and compared to the count of deer provided by TPWD. The initial scaling resulted in an overestimate in the number of deer in some areas and an underestimate in others. The

study site was subsequently split by ecoregion and NDVI was used to linearly scale density in the range 0–45 deer per km^2 in the Edwards Plateau and 0–15 deer per km^2 in the South Texas Brush ecoregions for both April (*LS3*) and December (*LS4*).

2.2.8. *Epidemic simulation model*

The same modeling scenario was used for all model comparisons: one cell was selected as infected to initiate the simulation and this cell was used as the starting point for all simulations. For every simulation of the model, each cell centroid was allowed to interact with other cells within a 2000 meter neighborhood. The model was simulated for a time period representing 100 days and 100 model runs were simulated for each dataset, yielding a total of 10,000 iterations. The median number of deer infected and median area affected (km^2) were used to characterize each set of simulations at the 100^{th} model day.

The population density, distribution, and habitat requirements of deer within the study area were explicitly incorporated in the model. We assumed the home ranges of deer in the study area were within a distance of 2 km and no interactions took place beyond this distance. The interaction probabilities between locations were weighted using a kernel defined by the inverse of the distance from the cell centroid, with the value being a fraction of a pre-specified bandwidth (1000m). The weights were reduced when neighbors were further away than the pre-specified bandwidth, and increased when they were closer.

In the model ("SIRCA"), deer could pass through four disease states: from susceptible to latent, from latent to infectious, from infectious to immune and finally back to susceptible. Parameter values for the latent, infectious and immune periods were based on the literature, predominantly laboratory based studies of FMD infection in deer [31, 35, 64, 96]. These transitions partially determined the dissemination rate of FMD between cells [33]. The first transition depended on contact rates between susceptible and infected deer cells in the previous time step. Homogenous mixing was assumed to take place within but not between cells.

The probability of interaction between neighboring locations also depended on the density of susceptible deer in the two locations, calculated as the product of their probabilities. Locations with more than a maximum threshold of deer were assigned a probability of 1.0. The remaining locations were linearly scaled into the interval 0 to 1 by dividing each location's density by the maximum threshold value [96]. To incorporate stochasticity into the model, interactions between a susceptible location and an infectious neighbor occurred when a random number from a pseudo-random number generator (PRNG) using the Mersenne Twister mtl19937 algorithm [60, 94] was below the assigned probability threshold for that pair of locations [96].

Once a cell was infectious the second, third, and fourth transitions in the model depended on the length of the latent, infectious and immune periods as assigned in the

model parameterization [31, 35, 64, 96]. The specific values were assigned randomly within the corresponding parameter ranges using a uniform distribution. The baseline model parameter values are summarized in Appendix A.

The geographic automata model framework is particularly suited to modeling foreign animal diseases in wild animal populations. Geographic variations are explicitly modeled in a simple manner and individual-level animal census data is not required, as long as an approximate statistical distribution is available [44]. In addition, the model does not require complex mathematical equations, but instead relies on local relationships between cells [44]. The assumption of local spread is a reasonable assumption for white tailed deer populations: in the absence of disturbance, deer are unlikely to move outside their local home range [96].

2.3. Data analysis

The results of the model simulations were analyzed using agglomerative clustering to identify groups of geostatistical methods that produce similar results at the 100^{th} model day. This was done using the agnes algorithm within the Cluster package in R [46, 76].

The similarity matrix used in the clustering analysis was developed by calculating the difference between each pair of geostatistical methods as the square root of the average squared difference between each pair of model runs:

$$similarity = sqrt(\frac{1}{n^2}\sum_{i=1..n}\sum_{j=1..n}(m_{1i} - m_{2j})^2)$$

Where n is the number of model runs (100 in this case), m_{1i} is the ith value for methodology 1, and m_{1j} is the jth value for methodology 2. Spatial risk maps of epidemic progression were generated using the number of times each unique location was infected across all simulations.

3. Results

3.1 *Predicted distribution and density of deer for each individual method*

Each of the estimation methods were compared with respect to the predicted number of deer per county as provided by TPWD. The number of deer in the study region predicted from each estimation method is summarized in Table 2. Estimates ranged from 385,939 to 768,493. In addition, the geostatistical methods resulted in different spatial representations of the distribution of deer in the study region. Examples of some of the distributions and the corresponding spatial risk maps are shown in Figures 3 and 4 and 5, respectively. Summary statistics for the predicted number of deer infected and area affected for each of the estimation methods (10,000 iterations each) is shown in Table 3. A graphic depiction of the median number of deer infected and the median area affected

by method is shown in Figure 6. Boxplots of the predicted outbreak distribution for each method are shown in Figure 7.

3.2. *Cluster analysis*

The clustering algorithm for the predicted distribution of deer infected identified two distinct clusters of methods, and one outlier (Figure 7). The first cluster included the *DC* and *DFB* methods, both of which were methods constrained within county boundaries. The second cluster included all other methods except *LS1*: the difference between this and other methods is apparent in Figure 3.

4. Discussion

Substantial differences in the estimated number of deer in the study region based on the geostatistical estimation procedure used were found: the total deer population ranged from 385,939 to 768,493. Substantial differences were also observed in the median predicted magnitude of the outbreak, which ranged from 1,563 to 8,896 deer infected. This variability in the predicted median outbreak size, as a result of using different geostatistical methods to describe the population at-risk, supports the argument that reporting only summary statistics from simulation models can be misleading. It is important that an attempt be made to consider the entire predicted outbreak distribution when summarizing modeling results, especially if these results are to be presented to policy-makers or to be used by decision-makers in the face of an outbreak.

Species predicted spatial distributions should be compared to known data, where available, and consideration should be given to how well the spatial estimate matches the species distribution reported from reputable data sources. Reports available from TPWD for this study region (for example, DC and DR, Fig. 3) indicate that the highest density of deer is found in the northern half of the study region [56]. Some of the spatial estimation methods better reflected this distribution than others. This is highlighted in Figure 3, where the predicted deer distribution for three of the methods is shown. *DC* predicts the highest density in the uppermost 3 counties, but fails to extend far enough south. *DR* appears to best represent the known density of deer in the study region, with the highest density in the northern half of the study region. *LS1* is clearly inconsistent with the deer estimate from TPWD, showing the highest density in the southwest portion of this region. Even though some of the spatial distributions do not accurately reflect the TPWD estimate, using these geostatistical methods still resulted in similar estimates of the total disease outbreak impact. Thus, the choice of geostatistical method for representing animal species distribution is probably secondary to the objectives of the study. If the aim is to estimate the overall impact of an FMD disease outbreak, results from this study suggest that the choice of geostatistical method is not critical. However, from the perspective of spatial analysis and predicting the likely spatial distribution of infected areas, the choice of geostatistical method becomes more important.

Study results indicate that the simpler methods for predicting white tailed deer density and distribution form one cluster, and the more complex methods form a second cluster (Figure 7). For these two clusters of methods, the mean predicted median outbreak size and median area affected were 1,599 deer and 110 km^2 and 4,578 deer and 263 km^2, respectively. Thus, broadly these two clusters resulted in a 2− to 3−fold difference in predicted outbreak size and distribution, indicating that the estimation methodology used to distribute deer has a significant impact on model predictions. Specifically in the present study, two representations of the population are necessary to capture the variability in the predicted outbreak size and distribution. The values in the dendrogram (Figure 7) are measures of the variation in the number of predicted infected locations. Cluster 2 values span a range of approximately 140. This characterizes the variability that one might expect in model predictions, depending on the method used to characterize the spatial distribution of an animal species of interest. Certain methods are closer together than others; for example *K1*, *K2*, *K4*, *LS2* and *LS4* are separated by a distance of only 70. The distance cut point used for cluster identification is somewhat arbitrary. For example, cluster 2 could be further subdivided if the variation of 140 is considered too large. This would result in the *DRU* method – a method that produced a distribution that was inconsistent with the TPWD report of deer distribution (highest densities in the southern portion of the study region) – being an outlier.

The underlying assumptions of the various geostatistical estimation methods should be considered, in addition to how well they predict the known data. Dasymetric mapping

assumes that the data (wildlife density and distribution over the landscape) has an underlying spatial pattern which can be characterized using ancillary data, such as habitat and carrying capacity. This assumption is reasonable for wildlife data. Dasymetric mapping methods (such as *DC*, *DFB* and *DRU*) that were applied within a political (county) or ecological (reporting unit) boundary further assumed that these ancillary attributes were captured within the bounds placed on the data. For these methods, it was assumed that habitat and carrying capacity attributes were adequately captured at the sub-county or reporting unit level. This assumption may not be entirely valid. We know that wildlife, especially white tailed deer, view and select habitat at the patch level and that patch dynamics do not necessarily follow political boundaries, that is, a suitable patch could easily cross county boundaries leading to an inaccurate estimate of the density or distribution.

Based on the deer distribution data available from TPWD, the reporting unit scale appears too coarse to adequately model deer distribution using the ancillary attributes of habitat and carrying capacity. In addition, methods that were forced to distribute within county boundaries lead to demarcation lines (horizontal bands) along these boundaries in the resulting spatial estimate of the species distribution. Although we know these boundaries are not realistic, they often represent the source of the only census data available.

Kriging has an underlying assumption of spatially continuous data. Wildlife distributions are typically not considered continuous and therefore kriging may not be the most appropriate method to use. Estimation methods that depended on remotely sensed imagery (*LS1 – LS4*) assume there is a relationship between NDVI values and deer density and distribution. NDVI measures vegetative greenness, and it was assumed that there is a linear relationship between deer density and NDVI value. However, this assumption has not been validated. The distribution derived from NDVI did not always adequately characterize the known deer distribution. For example, the outbreak distribution produced by the *LS1* method (Figure 7) was an outlier in the cluster analysis. How this method might be applied requires further investigation.

Demarcation lines in the data are an artifact of the artificial (administrative and political) boundary that is placed on the data (county or reporting unit). Dasymetric mapping methods create a statistical deer distribution designed to remove the effect of artificial boundaries [17, 54, 75]. However, when these methods are used within a boundary the resulting distribution tends to suffer from demarcation at that boundary. Methods that do not explicitly include boundaries in the estimation procedure (*DR*, *K1*, *LS2*) did not suffer from demarcation in the resulting estimated distribution. Because we are interested in modeling disease spread in a multi-county region, a clear demarcation line (for example, high density to zero or extremely low density across a single 1 km^2 pixel) presents a problem for epidemic modeling. Given that geographic automata models operate at the local level, the distribution and density of surrounding cells is very

important in determining whether and how the disease will spread. An ideal method is one that results in both realistic and suitable spatial animal distributions for spatial modeling of disease spread.

The results of this study demonstrate that to eliminate demarcation in the spatial distribution data at the level of aggregation available it is necessary to use a regionalized interpolation method (kriging) or a method involving individual pixel level data (NDVI). However, these methods that smooth population distributions result in much larger estimates of the magnitude of the outbreak and the spatial distribution of infection (Figure 4). The actual estimate of the overall population density appears to play little, if any, role in the resulting magnitude of the predicted outbreak. The distribution and, more specifically, the smoothness and spatial continuity of the distribution appear to have a major role in the predicted outbreak size (Figure 4). This is highlighted in Figures 4, 6, 7 and Table 3. The group with low spatial continuity: *DC, FS1 – FS3, DR, DRU, DFB* and *LS4* all have zeros in their distributions where outbreaks failed to occur. Their fifth percentiles (Table 3) are all zero, indicating that outbreaks did not occur in at least 5% of the model runs. All of the surfaces with low spatial continuity had zeros in their fifth percentiles for 98 of the 100 runs indicating that in only 2 of the 100 runs did an outbreak start. The group with high spatial continuity: *K1 – K4* and *LS1 – LS3* all have much higher values for their fifth percentiles indicating that an epidemic always occurred for these surfaces. This result is to be expected given the spatial formulation of the model. The more continuity in the spatial distribution, the greater the opportunity for

interactions between locations and therefore more interactions will occur even when there are lower interaction probabilities.

The need to use spatially-explicit models to simulate the spread of FMD has been recognized [33, 45], and spatial heterogeneity has been identified as possibly the greatest challenge to realistically representing FMD spread through a landscape [22]. In addition to capturing the spatial heterogeneity of the population across the landscape, wildlife distributions need to be seasonally-dynamic, since these species are particularly affected by variations in climate and natural resources. Such temporal dependency may have a significant impact on the spread of disease within wildlife populations, and further, into domesticated animal populations of interest [21]. Temporal dependency should be incorporated in future studies of disease spread in potential wildlife reservoirs. While we have included some level of temporal dependency with the NDVI surfaces, a more detailed analysis is necessary in the future.

The model used in this study has been used previously to investigate wildlife-domestic species interactions between feral pigs and cattle [21, 96] and between wild deer and cattle [96]. In the current study, our focus was on the potential spread of FMD in wild deer populations. We made the simplifying assumption that because of relatively low grazing densities in this extensively management livestock system, cattle do not contribute greatly to disease spread. Also, we focused on the initial stages of disease spread (≤100 days); assuming a minor role for domestic livestock during this initial

phase of an outbreak is likely to be valid. The duration of resistance to FMD virus reinfection was assumed to be 90–180 days. Although this assumption may be unrealistically low, it probably had little impact on study results because of our focus on the initial stages of disease spread.

The model predictions are likely to be sensitive to temporal fluctuations in the population densities (for example, seasonal or annual population trends, particularly if these fluctuations occur differentially across geographical areas of the study region. Thus, study results should be viewed as the average effect of different representations of animal densities. More research is needed to determine if the methods of representing animal densities, or temporal fluctuation of those densities, are more important in determining the outcome of a disease incursion such as FMD. Care should be exercised when using the same epidemiological parameters on different spatial landscapes. This is even more problematic because epidemiologic parameters are estimated from a disease outbreak that occurs within a given spatial landscape. Given that FMD has not occurred in the U.S. since 1929, it is virtually impossible to estimate the epidemiologic parameters, should FMD virus be introduced into the deer population. However, the model system does incorporate uncertainty by using parameter ranges [96]. Regarding the role of spatial heterogeneity on parameter estimation, we feel that the model is robust even in the absence of detailed parameter estimates. Spatial heterogeneity has been implicitly included in the model by the use of density to adjust disease transmission. Furthermore, by using landscape variability (key habitat features) in the distribution

methodologies and density to control interaction in the simulation model, we have incorporated heterogeneity of transmission via a “self-adjusting” model that varies across the landscape. We have captured variation in both the distribution of susceptible hosts and contact rates over the landscape: this is the primary underlying cause of the differences between model results.

CHAPTER III

CRITICAL PARAMETERS FOR MODELING THE SPREAD OF FOOT AND MOUTH DISEASE IN WHITE TAILED DEER POPULATIONS

Modeling potential disease spread in wildlife populations is important for predicting, responding to and recovering from a foreign animal disease incursion. To make epidemic predictions using a simulation model, a number of important (and often unknown) parameters must be estimated. A series of simulation experiments were conducted to determine how estimates of the latent and infectious period, number of neighbors (contacts) and population size impact the predicted magnitude and distribution of foot and mouth disease (FMD) outbreaks in white tailed deer in southern Texas. Outbreaks were simulated using a previously developed and applied susceptible-infected-recovered geographic automata model. The study region was a 9-county area (24,000 km^2) of southern Texas. The magnitudes and distributions of the predicted outbreaks were evaluated by comparing the median number of deer infected and the median area affected (km^2), respectively, and with spatial risk maps of epidemic progression. There were substantial differences in the estimated predicted number of deer and locations infected, based on the model parameters used (range: 3,779-119,879 deer infected and 227-6,526 locations affected). There were also substantial differences in the spatial risk of infection based on the model parameters used. The predicted spread

of FMD was found to be most sensitive to the latent period and the number of neighbors, based on >10% change from the baseline scenario predicted median number of deer infected and number of locations affected. How these parameters are estimated is likely to be critical in studies on the impact of FMD spread in situations in which wildlife reservoirs might potentially exist.

1. Introduction

The aim of this research is to evaluate the sensitivity of epidemic model predictions to disease spread parameters estimated in wildlife species. Specifically this chapter evaluates the effect of the latent and infectious periods, the number of neighbors (contacts) and local-level population density on model predicted size and distribution of foot and mouth disease (FMD) outbreaks in white tailed deer. This research provides critical insight into the impact that these estimated parameters have on modeling predictions. It is important because model predictions may be used to guide policy and evaluate mitigation strategies prior to an outbreak [86, 96]. In addition, modeling may be used during an outbreak to inform response strategies, particularly disease mitigation strategies in wildlife populations. To assess these issues, this paper addresses the impact of various estimates of important disease parameters on the predicted outbreak distribution of FMD in white tailed deer in southern Texas.

FMD is a highly contagious viral disease of cloven-hoofed animals, affecting both domestic and wild Artiodactyla species, including deer. Deer have been infected both naturally and experimentally [31, 35, 64, 74], and deer-to-deer and deer-to-cattle transmission has been observed [35]. Experimentally infected white tailed deer exhibited intermediate disease severity compared with susceptible livestock species (such as cattle, sheep and goats) and approximately 10% of those infected in a 1924 outbreak in California displayed typical signs of FMD infection [64]. The United States has been free of FMD since the 1929 outbreak.

During the 2001 FMD outbreak in the United Kingdom, it was feared that a number of the deer species in the country (red, fallow) might become infected and potentially act as a reservoir for the disease [16, 85]. A similar concern was also expressed in the Netherlands during the 2001 FMD outbreak [27, 85]. However, evidence of infection in deer was not observed in either of these more recent outbreaks [27].

In areas of the United States where livestock are extensively grazed, the potential for interaction and contact with susceptible wildlife species, such as white tailed deer, is high [96]. Deer traverse and forage in fields between farms, and enter premises containing animal feed and slurry [85]. Additionally, supplemental feeding of white tailed deer for hunting purposes is common, potentially leading to increased contact [96]. Given the widespread distribution of wildlife species susceptible to FMD virus infection and the potential for interaction with livestock, modeling the spread of the

disease in wildlife populations is an important resource in our ability to predict, respond to and recover from a foreign animal disease incursion.

To model the spread of FMD in a wildlife population, such as white tailed deer, estimate of a range of disease and spatial parameters is critical. The distribution of the species of interest must be estimated spatially prior to parameterizing a disease spread model and simulating disease spread [37]. Once the population distribution has been described, disease parameters such as the latent and infectious periods must be estimated prior to modeling disease spread. In addition, the number and type of contacts both within and between species must be estimated. Unfortunately, the values of these parameters in wildlife are usually unknown. Laboratory studies have been used to estimate the period of latent infection and the length of the infectiousness of various species [31, 35, 64, 74]. While these parameters are the "best" estimates available, they may not accurately capture the dynamics of the disease in the field. Given the uncertainty surrounding the parameter values, probability distributions are often used to model the parameters for disease spread. These distributions might be based on little information, such as informed "guesses" of the likely minimum and maximum parameter values. Sensitivity analysis can be used to identify parameters to which the model is particularly sensitive and for which better data should be sought.

Epidemics have historically been modeled using differential equations [1, 24, 84]. However, differential equation models do not directly address the local character of

disease spread or complex boundary conditions [84]. Geographic Automata (GA), generalizations of cellular automata models, are capable of handling non-tessellated data (points). Both cellular and geographic automata provide an alternative to differential equation based epidemic spread models. They treat time as discrete and interactions as localized [84] and have been applied to a wide range of disease spread problems [1, 8, 23, 30, 43, 78, 95]. Susceptible-infected-recovered models have been built into Geographic Automata to examine the spatial and temporal propagation of epidemics [8, 29, 30, 43, 53, 78, 84]. However, this approach has rarely been used to model the spread of infectious diseases in wildlife populations. The influence of spatial estimation techniques on the predicted spread of FMD in white tailed deer using a geographic automata model has been explored [37]. However, the effect of estimated disease related parameters on model predicted spread of FMD in white tailed deer populations has not been evaluated.

The objectives of this study were: 1) To apply a range of values to critical disease parameters in the geographic automata model; 2) Describe the predicted FMD outbreak distribution that might be observed, given the various estimates used; and 3) Compare the predicted FMD outbreak distributions for each of the parameters varied.

2. Materials and Methods

2.1. *Study site*

The study site, a nine county area of southern Texas bordering Mexico (Figure 1), has been previously described [37, 96]. It consists of two Ecoregions, the Edwards Plateau (EP) in the north and the South Texas Brush (ST) in the south. Seasonal variation in the overall study region is characterized by hot, dry summers and mild, moist winters, with average annual rainfall ranging between 750 and 1200 mm. The EP Ecoregion is predominately rangeland and is home to the highest concentration of deer in Texas, with an estimated 100 deer per 405 hectares [88].

The ST Ecoregion is considered a brush community. White tailed deer hunting has increased in this Ecoregion and the vegetation is actively managed to support hunting [88]. Population densities of white tailed deer in this Ecoregion are considered moderate with an estimated 29 deer per 405 hectares [88].

2.2. *Data source*

The estimated distribution of deer used to represent the deer population for the baseline scenario was previously derived and is described in Chapter 1 of this dissertation [37]. Deer densities were represented as points (centroids) for all modeling scenarios.

2.2.1. *Baseline scenario in the epidemic simulation model*

The same model initiation procedure was used for all model comparisons: one location was selected as infected to initiate the simulation and this cell was used as the starting point for all simulations. The identical spatial location was used for the latent and infectious periods and number of neighbors. This location corresponds to the index location used in a previous study of the sensitivity of the model to spatial estimates of deer distribution [37]. A different spatial location for the index infection was used to evaluate the impact of both global and local population density. This selected location corresponds to the index location for seasonal spread of disease and policy and mitigation shown in Chapters IV and V of this dissertation. The change in index location for global and local population density was motivated by the need to incorporate both a high density and low density index herd for comparison purposes. For every simulation of the model, each location was allowed to interact with other points within a 2000 meter neighborhood. The model was simulated for a time period representing 100 days and 100 model runs were simulated for each dataset, yielding a total of 10,000 iterations. The median number of deer infected and median area affected (km^2) were used to characterize each set of simulations at the 100^{th} model day.

The population density, distribution, and habitat requirements of deer within the study area were explicitly incorporated in the model. As a baseline, we assumed the home ranges of deer in the study area were within a distance of 2 km and no interactions took place beyond this distance. The interaction probabilities between locations were

weighted using a kernel defined by the inverse of the distance from the cell centroid, with the value being a fraction of a pre-specified bandwidth (1000m). The weights were reduced when neighbors were further away than the pre-specified bandwidth, and increased when they were closer.

In the model, deer could pass through four disease states: from susceptible to latent, from latent to infectious, from infectious to immune and finally back to susceptible. Baseline parameter values for the latent, infectious and immune periods were based on the literature, predominantly laboratory-based studies of FMD infection in deer [31, 35, 64, 74]. These transitions partially determined the dissemination rate of FMD between locations [33]. The first transition depended on contact rates between susceptible and infected deer locations in the previous time step. Homogenous mixing was assumed to take place within but not between locations.

The probability of interaction between neighboring locations also depended on the density of susceptible deer in the two locations, calculated as the product of their probabilities. Locations with more than a maximum threshold of deer were assigned a probability of 1.0. The remaining locations were linearly scaled into the interval 0 to 1 by dividing each location's density by the maximum threshold value [96]. To incorporate stochasticity into the model, interactions between a susceptible location and an infectious neighbor occurred when a random number from a pseudo-random number

generator (PRNG), specifically the Mersenne Twister mt19937 algorithm [60, 94] was below the assigned probability threshold for that pair of locations [96].

Once a cell transitioned to infectious the second, third, and fourth transitions in the model depended on the length of the latent, infectious and immune periods as assigned in the model parameterization [31, 35, 64, 74]. The specific values were assigned randomly within the corresponding parameter ranges using a uniform distribution. The baseline model parameter values are summarized in Appendix A.

The geographic automata model framework is particularly suited to modeling foreign animal diseases in wild animal populations. Geographic variations are explicitly modeled in a simple manner and individual-level animal census data is not required, as long as an approximate statistical distribution is available [44]. In addition, the model does not require complex mathematical equations, but instead relies on local relationships between cells [44]. The assumption of local spread is a reasonable assumption for white tailed deer populations: in the absence of disturbance, deer are unlikely to move outside their local home range [96].

2.2.2. *Population density scenarios*

The density of deer at each spatial location (centroid) was increased and decreased by 10% respectively, resulting in two additional datasets which were simulated using the baseline model parameters specified above.

2.2.3. *Latent period*

The latent period uniform probability distribution was varied using three sets of parameter ranges: 1−5 days, 3−5 days (baseline) and 5−10 days. The actual latent period for each location (centroid) was randomly sampled from a uniform distribution using the above ranges.

2.2.4. *Infectious period*

The infectious period uniform probability distribution was varied using three sets of parameter ranges: 1−14 days, 3−14 days (baseline) and 14−28 days. The actual infectious period for each location (centroid) was randomly sampled from a uniform distribution using the above ranges.

2.2.5. *Neighbors*

The number of neighbors that a given infected location (centroid) was allowed to interact with at each time step was varied to represent 1st through 3rd order neighborhoods for each infectious location. A 1000 meter neighborhood utilizing the 4 nearest neighbors, a 2000 meter neighborhood (baseline) representing 12 nearest neighbors and a 3000 meter neighborhood representing 28 nearest neighbors were used to simulate spread over a varying landscape area.

2.2.6. *Reduced population density within a local neighborhood*

The impact of local population density reduction was evaluated by reducing the density of locations (centroids) within a 10 kilometer distance from each of two selected initiation points (high density and low density). Within the 10 kilometer neighborhood from each index location, densities were reduced by 10% to 50% in 10% increments, yielding 10 additional datasets for model comparison.

2.3. Data analysis

The predicted spread of FMD was characterized for each set of parameters using the median number of deer infected and the median land area affected, together with 5^{th} and 95^{th} percentiles, interquartile range (IQR) and skewness and kurtosis. Sensitivity of the model to the parameter ranges was assessed by calculating a percent change from baseline. In addition, the predicted spatial distribution of disease spread was evaluated using spatial risk maps. Spatial risk maps were created by calculating the probability of infection across 100 iterations of the geographic automata model for each spatial location affected.

3. Results

3.1 *Predicted distribution and density of deer for each parameter*

Summary statistics for the predicted number of deer infected and land area affected for each of the estimated parameters (10000 iterations each) are shown in Tables 4 through

15. Spatial risk maps for each of the estimated parameters are shown in Figures 9 through 16. The model was found to be sensitive to a >10% change compared to the baseline scenario predicted number of deer infected and locations infected for the parameter ranges used for the latent and infectious periods, the number of neighbors (contacts) and the population density both at a global and local level. Variation in the latent period affected the model predicted spread of FMD (Table 4 and 5). A higher range of sampled values (5−10 days) resulted in a 90% decrease in the median predicted spread (3779 infected deer) compared to the baseline latent period (3−5 days) parameter (38537 infected deer). A 89% decrease in the median number of infected locations was also observed for the higher latent period range versus baseline (227 versus 1985, respectively). A lower range of sampled values (1−5 days) resulted in a 106% increase in the median predicted spread (79242 infected deer) versus baseline. A 108% increase in the number of infected locations (4123) was observed. The spatial pattern of infection was also sensitive to the latent period range (Figure 9). A shorter latent period resulted in a slightly larger core area of spread versus baseline (>50% risk) but in a small proportion of model runs (<20%) there was a much larger area of spread. A long latent period resulted in a much smaller spatial distribution of infection in all risk categories (10 – 100%; Figure 9).

Variation in the infectious period also affected the median model predicted spread of FMD for the lower range of sampled values, compared to the baseline (Table 6 and 7). However, no difference was observed between the higher range of sampled values and

the baseline (Table 6 and 7). A lower range of sampled values (1−14 days) resulted in a 66% reduction in both the median predicted spread of FMD (13063 versus 38537 deer infected, respectively) and the predicted number of infected locations (679 versus 1985, respectively), compared to baseline. A higher range of sampled values (14−28 days) resulted in a <10% difference in the median predicted spread based on either number of deer infected (36829 versus 38537, respectively) or number of infected locations (2114 versus 1985 locations infected, respectively), compared to the baseline infectious period parameter. The spatial pattern of infection was also sensitive to the infectious period range (Figure 10). A short infectious period substantially reduced the spatial risk of infection for all risk categories (10−100% risk; Figure 10), whilst a long infectious period resulted in a slight increase in the risk of infection for the core area (>50%) compared to the baseline infectious period, particularly in the southern portion of the affected area.

Variation in the number of neighbors (contacts) affected the model predicted spread of FMD (Table 8 and 9). A higher number of neighbors (28) resulted in a 211% increase in the median predicted spread of FMD (119873 infected deer) compared to the baseline number of 12 neighbors (38537 infected deer) and in a 229% increase in the number of infected locations (6526) versus baseline (1985). A lower number of neighbors (4) resulted in a 91% decrease in the median model predicted spread (3606 infected deer) and a 90% decrease in the number of infected locations (205), versus baseline.

Variation in the global population density significantly affected the model predicted spread of FMD (Table 10 and 11). An increase of 10% of the overall population density resulted in a 27% increase in the median predicted spread (48773 deer infected) versus baseline (38537 deer infected). A 15% increase was observed in the number of infected locations (2228) versus baseline (1985). A global population decrease of 10% resulted in a 24% decrease in the median predicted spread of FMD (29177 infected deer) compared to the baseline population density. A 17% decrease was observed for the number of infected locations (1650) versus baseline. The spatial pattern of infection was also sensitive to the global population density (Figure 12). A reduced global population density resulted in a smaller core area of infection (>50% risk), compared to the baseline, whilst an increased global population density resulted in a larger core area of infection.

Variation in the local population density (within 10km proximity to the index case) also affected the model predicted spread, compared to the baseline scenario (Table 12 through 15). A decreasing local population density, varied from 10−50% reduction within 10 kilometers of the high density index case, resulted in decreased median predicted spread and number of infected locations for most scenarios (Table 12 and 13). A 10% decrease in local population density resulted in almost no difference in the predicted median number of deer infected (52674) or locations affected (2505) versus baseline (56092) locations. A 20% reduction in local population density resulted in an 11% decrease in the median predicted number of deer infected (50082) and a 9%

reduction in the number of infected locations (2413), compared to baseline. A 30% reduction in local population density resulted in a 18% decrease in the median predicted number of deer infected (45926) and an 15% reduction in the number of infected locations (2223), compared to baseline. A 40% reduction in local population density resulted in a 31% reduction in the median predicted number of deer infected (38901) and a 27% reduction in the number of infected locations (1934), compared to baseline. A 50% reduction in the local population density resulted in a 51% decrease in the median predicted number of deer infected (27424) and a 46% decrease in the number of infected locations (1435), compared to baseline. For all scenarios, disease spread was observed in 100% of the simulations (Table 12 and 13). The spatial pattern of infection was also sensitive to the local population density (Figure 13 and 14). An increasing reduction in the core area of infection (>50% risk) is shown for all risk categories (10-100%) of reduced local density along with corresponding increases in the low probability categories.

A decreasing local population density, varied from 10−50% reduction within 10 kilometers of the low density index case, resulted in decreased median predicted spread and number of infected locations for all scenarios (Table 14 and 15). A 10% reduction in local population density resulted in a 32% decrease in the median predicted number of deer infected (4315) versus baseline (6357), and a 30% reduction in the median number of infected locations (413) versus baseline (590). A 20% reduction in the local population density resulted in a >99% decrease in the median predicted number of deer

infected (10) and in the median number of locations infected (2), versus baseline. A 30% reduction in local population density resulted in a >99% decrease in the median predicted number of deer infected (8) and a >99% decrease in the number of locations infected (2). A 40 and 50% reduction in local population density resulted in equal reductions in the predicted median number of deer infected and location infected. Both scenarios resulted in nearly a 100% decrease in the predicted median number of deer infected (3) and the predicted number of locations infected (1), versus baseline. For all scenarios, disease spread was observed in all simulations (Table 14 and 15). The 40 and 50% reduction scenarios resulted in right skewed distributions, indicating that the level of disease spread for these scenarios was low for almost all runs with a few larger outbreaks. However, even the "large" outbreaks for these distributions were smaller than those observed for the baseline (65 and 878 infected deer respectively, versus 11362 for the baseline). This is also shown in the spatial pattern of spread (Figure 15 and 16). The core area of infection (>50% risk) is almost completely absent after a 20% reduction in the local population density. The overall spatial risk of infection is also drastically reduced as the local population density decreases. For the 50% reduction in local population density, the risk of infection for almost all spatial locations is reduced to less than 20%.

4. Discussion

We found that the model is sensitive to the parameters examined: latent period, infectious period, number of neighbors, and global and local population density. A short latent period produced a >100% increase in the model predicted spread for both the number of deer infected and locations infected. A long latent period resulted in 90% decrease in the predicted median number of deer infected and a 89% decrease in the median predicted number of locations infected. A short latent period resulted in increased disease spread due to the interaction between the short period of latency (1 – 5 days) and a fairly long period of infectiousness (3 – 14 days). These parameters increased the probability of interaction between susceptible and infectious locations in the model. Fundamentally, the short latent period allowed for a faster progression of the infection over space. This interaction between a short latent period and a longer infectious period has been previously shown to increase disease spread using a cellular automata model [21]. Conversely, a long latent period resulted in what appears to be a "burn out" effect. By the time each location transitioned to infected (after 5 – 10 days), most of its neighbors were already latently infected due to contact with a shared source of infection (with an infectious period of 3 – 14 days). Essentially, there was an interaction between a long latent period (almost equal to the length of the infectious period) and the neighborhood structure of the data. When all locations were at 1km resolution the contact neighborhoods were very similar for all infectious cells, allowing for very few locations that were available to infect as each location makes its transition

from latent to infectious. A short infectious period reduced both the model predicted median number of deer infected and the number of location infected by 66%. A long infectious period had almost no effect on the model predicted spread (<10% percent change from baseline). The spatial risk of infection was also sensitive to a short infectious period. A short infectious period resulted in a different spatial pattern of infection risk than either the baseline or longer infection periods. This indicates that with a short infectious period the disease does not have the ability to progress over a very large spatial area: the lower range of the infectious period (1−2 days) effectively stops the spread of infection, compared with the baseline (minimum 3 day infectious period).

The model is also sensitive to the number of neighbors (contacts). A higher number of neighbors resulted in a 211% increase in the median predicted spread of FMD and a 229% increase in the predicted number of infected locations, compared to the baseline number of neighbors (12). A lower number of neighbors resulted in a 90% decrease in the median model predicted number of deer infected and in the predicted number of infected locations. This result is consistent with previous sensitivity analyses using this model [90], and it makes biological sense because as the number of susceptibles that come into contact with infected locations increases, it is expected that disease spread will also increase.

The model of disease spread in white tailed deer appears to be more sensitive to the assumed length of the latent period than the assumed length of the infectious period.

This impact of the latent period assumed has been shown in previous sensitivity analyses of the SIRCA model [21]. A short latent period allows the disease to spread more quickly through the population by increasing the probability of interaction between infectious and susceptible locations. Other studies have found that an increased infectious period leads to increased disease spread, however the parameter ranges used and the species modeled are different than those in the present study [21]. It is possible that in areas where deer densities are high (such as in the Northern portion of the study area), all of the susceptible neighbors have been infected by the 14th day of the infectious period. Thus, increasing the period of infectiousness to 28 days has little impact on disease spread. This indicates that there might be a threshold value for the period of infectiousness, given the specific population density and size of the neighborhood for each location (assuming the length of the infectious period is shorter than the immune period). The effect of the infectious period and the possibility of a threshold value require further study. In this study, each location could contact up to the 12 nearest neighbors within a distance of 2km in the baseline scenario. If the size of the neighborhood were increased together with the infectious period, the results would likely show increased spread. Whether such an increase is additive or multiplicative needs to be investigated in future research. In addition, future research should examine this effect for a lower density region such as the southern region of the study area.

The model appears to be sensitive to both the global and local population density. Shifts in the global population density of 10% (increase and decrease) at each spatial location

resulted in a ~25% increase or decrease in the median predicted number of deer and a ~15% increase or decrease in the predicted number of locations infected, respectively. In both cases, the predicted number of deer and locations infected was sensitive to population density. This result is not surprising given the formulation of the model. Density is used to adjust the probability of contact between locations; so as the density increases or decreases, the likelihood of contact between locations is adjusted accordingly. The effect of the change in density at the local level appears to have a greater effect in lower densities areas (in the current study, simulated incursions in the northern versus southern region of the study area; Figures 13-16). This is likely due to the fact that in very high deer density areas there is also a high level of spatial contiguity; therefore a reduction in the density does not have the same impact as it does in lower density areas. The assumed biological relationship between density and contact needs to be better characterized in wildlife species that might act as reservoirs of FMD disease.

The sensitivity of the model to local population density was investigated for both a high and low density index location. For a high density index location, the population had to be reduced substantially more than for the low density index location to achieve similar levels of reduced spread of disease. Decreasing local population densities from 10 to 50% yielded a decrease in the predicted median number of deer infected of 6%, 11%, 18%, 31% and 51%, respectively, and a decrease in the predicted number of infected locations of 5%, 9%, 15%, 27% and 46%, respectively. While the area of the core spatial spread (>50% risk) was reduced for each level of local density reduction, the overall

distribution of spatial risk was relatively constant. This indicates that, in some situations, the spatial spread of disease would not be greatly different even with a 50% reduction in the local population density.

For the low density index location, the model is sensitive to the change in the density. Decreasing local population densities from 10 to 50% yielded a decrease in the median number of deer infected of 32% for a 10% decrease in density and a >99% decrease in the median number of deer infected for all population reductions >10%. A decrease in the predicted number of infected locations of 30% was observed for a 10% decrease in density, while a >99% decrease was observed for 20-50% density reductions. The spatial pattern of disease spread was also substantially reduced as local population density was decreased. The core area of infection (>50% risk) was almost completely absent after a 20% decrease in the local population density. This finding has potentially important policy implications: in areas of lower density white tailed deer populations, local population density reduction could be an effective strategy to reduce disease spread either prior to or during an outbreak. Future research considering local population density reduction as a potential mitigation strategy to prevent disease spread in white tailed deer should be considered.

The model used in this study has been used previously to investigate wildlife-domestic species interactions between feral pigs and cattle [21, 96], between wild deer and cattle [96] and to evaluate the impact of spatial estimates of deer distribution [37]. In this

current study, our focus was on the potential spread of FMD in wild deer populations. As in the previous study [37], the simplifying assumption was made that cattle do not contribute greatly to disease spread because of relatively low grazing densities in this extensively management livestock system. Also, the study focused on the initial stages of disease spread (≤100 days). Therefore, assuming a minor role for domestic livestock during this initial phase of an outbreak is likely to be valid. The duration of resistance to FMD virus reinfection was assumed to be 90–180 days. Although this assumption may be unrealistically low, it probably had little impact on study results because of the focus on the initial stages of disease spread. Caution should be exercised when using the same epidemiological parameters on different spatial landscapes. This is even more problematic when epidemiologic parameters are estimated from a disease outbreak that occurs within a given spatial landscape. Given that FMD has not occurred in the U.S. since 1929, it is virtually impossible to estimate valid epidemiologic parameters, should FMD virus be introduced into the deer population. However, the model system does incorporate uncertainty by using parameter ranges [96]. The model is robust even in the absence of detailed spatial heterogeneity parameter estimates: spatial heterogeneity has been implicitly included in the model by the use of density to adjust disease transmission. Furthermore, by using landscape variability via key habitat features in the distribution methodologies and density to control interaction in the simulation model, heterogeneity of transmission has been incorporated via a “self-adjusting” model that varies across the landscape. Variation in both the distribution of susceptible hosts and

contact rates over the landscape has been captured: this is the primary underlying cause of the differences between model results.

This is the first study to define the range and distribution of estimates of outbreak magnitude generated by various estimates of critical model parameters (both aspatial and spatial) for FMD spread in white tailed deer.

CHAPTER IV

THE IMPACT OF SEASONAL VARIABILITY IN SOUTHERN TEXAS WHITE TAILED DEER POPULATIONS ON THE PREDICTED SPREAD OF FOOT AND MOUTH DISEASE

Modeling potential disease spread in wildlife populations is important for predicting, responding to and recovering from a foreign animal disease incursion. Wildlife species are heavily influenced by their environment, and seasonal fluctuations in population distributions might impact disease spread and therefore epidemic control. We conducted a series of simulation experiments to determine how seasonal estimates of the spatial distribution of white tailed deer impact the predicted magnitude and distribution of potential foot and mouth disease outbreaks in south Texas. Outbreaks were simulated using a susceptible-infected-recovered geographic automata model ("SIRCA"). The study region was a nine county area (24,000 km^2) of southern Texas, comprising 2 distinct Ecoregions. Seasonal deer distributions were estimated using spatial autoregressive lag models and a previously developed baseline deer distribution as the dependent variable and an averaged normalized difference vegetative index value as the independent variable for each season. The magnitude of the predicted outbreaks for each of the 4 seasons and 2 Ecoregions were evaluated by comparing the median number of deer infected and median number of spatial locations infected, respectively. A non-

parametric Kruskal-Wallis test was used to test for differences in predicted epidemic spread in the 8 treatment groups (Ecoregion and season). Miller's multiple comparison procedure was used to determine groupings in the predicted epidemic spread. Substantial differences were observed in the median predicted magnitude of the FMD outbreak both by season and Ecoregion: the number of deer predicted to be infected ranged from 7,792 to 19,493. Results suggest that the outcome of an FMD incursion in a population of wildlife, such as white tailed deer in south Texas, might depend on both where and during which time of year the incursion occurs.

1. Introduction

The aim of this research is to incorporate the effect of seasonal variability of deer distributions, using the normalized difference vegetation index (NDVI) as a measure of forage availability, into predictions of the potential spread of foot and mouth disease (FMD) virus in deer populations in southern Texas. Specifically, this chapter aims to generate an estimate of the density and distribution of deer for each of four seasons in two Ecoregions, and subsequently, to model the potential seasonal spread of FMD in the white tailed deer population within the study area. Seasonal and temporal distribution of species has been identified as one of the critical factors limiting realistic modeling of infectious disease spread [96]. This research will provide insights into the impact that the estimated seasonal spatial distribution of populations has on modeling predictions and hence, potential implications for FMD response and mitigation strategies and policy.

There are an estimated 395,000 white tailed deer in south Texas, an area comprising of approximately 24,500 square km and 9 counties (Figure 1). White tailed deer represent an important financial resource to a substantial number of ranchers in south Texas [14], and the deer population is actively managed for hunting and recreation purposes [14, 89]. Population management for optimum carrying capacity is important for maintaining herd levels and nutritional status [97]. Deer in the study region are primarily browsers (consuming leaves and twigs from shrubs and trees) during the fall [79]. Grasses and forbs have been found to be important dietary components during the spring [28, 62, 50]. Deer will only consume grass when it is tender and green (young), as deer can not digest mature grass [79]. Forb production in the study region is highly dependent on rainfall and season; forbs tend to be unpalatable to deer during late summer and late winter [79]. Given this shift in dietary habits, deer distributions are expected to vary by season, specifically based on rainfall and forage availability.

The NDVI is one of a number of vegetative indices derived from remotely sensed imagery. It is calculated from measured brightness values based on the absorption, transmittance and reflectance of energy by vegetation in the red and near-infrared portions of the electromagnetic spectrum [18, 58, 41]. The NDVI value is calculated from the near infrared (NIR) and visible red wavelength values as: (NIR – red) / (NIR + red) [39]. NDVI is associated with photosynthetically active radiation, and is the most commonly used index used to estimate vegetative growth [52].

NDVI data are collected by the National Oceanic and Atmospheric Administration Advanced Very High Resolution Radiometer (NOAA AVHRR) satellite at a spatial resolution of 1km and are available in a number of formats from the United States Geological Survey (USGS), National Mapping Division's Earth Resources Observation and Science (EROS) Data Center, including weekly and bi-weekly composites for the U.S. and 10 day global composites [82]. NDVI data have moderate spatial resolution (1km) but have high temporal resolution as the entire globe is imaged twice a day [82]. Bi-weekly maximum NDVI composites are created by using the maximum observed value for each composite period to reduce cloud contamination [25]. NDVI images are registered to the Lambert Equal Area Azimuthal map projection to ensure spatial accuracy to within 1 pixel ($1km^2$) [82]. The NDVI has been used in numerous studies on the classification of land use and temporal vegetation variability (onset, peak, senescence) [38, 57, 82, 92, 93]. The USGS has also conducted vegetation assessments using NDVI to monitor seasonal growth patterns in rangelands, forests and agricultural areas [26]. The NDVI has furthermore been used in studies of vegetation response to precipitation [20, 55, 98, 42].

A few studies have examined the relationship between the NDVI and stocking rate of livestock in the U.S. [70, 39]. A study by Showers et al. [83] conducted in north central Texas (Central Rolling Red Plains Major Land Resource Area) examined the relationship between NDVI value and diet quality of white tailed deer. It was found that

the NDVI, when used within season, was highly correlated with dietary measurements of deer for winter and spring (R^2>0.7). The correlation between the NDVI and deer dietary measurements during summer was weak (R^2=0.25). It is important to note that the authors only evaluated the NDVI in 3 seasons, using the following classifications: Winter (January – March), Spring (April – June) and Summer (July – September). Data for October through December were not evaluated. Marshall et al. [59] studied habitat use by mule deer in the desert southwest and evaluated the NDVI as a predictor for deer distribution. These authors found that the NDVI was significantly (P<0.05) associated with deer distribution in 3 of 4 seasons. No significant association was found in winter, and this was attributed to uniformly high quality forage availability at that time of year.

The need to use spatially-explicit simulation models for FMD has been recognized [33, 45] and spatial heterogeneity has been identified as perhaps the greatest challenge to representing FMD spread across the landscape [22]. Wildlife species are particularly affected by variations in climate and natural resources [96, 37]. To capture spatial heterogeneity across the landscape, wildlife distributions should therefore be seasonally-dynamic. Such temporal dependency may play an important role in the spread of disease within wildlife populations, and further, into domesticated animal populations [21]. The model used in this study has been previously described [21, 37, 96] and has been used in these previous studies to investigate wildlife-domestic species interactions between feral pigs and cattle [21, 96] and between wild deer and cattle [96] and within wild deer [37].

The objectives of this study were: 1) To incorporate seasonal fluctuations into the predicted distribution of deer in the study region by using bi-weekly composite NDVI values as a measure of forage availability in a regression model and 2) Describe the predicted FMD outbreak distribution that might be observed, given the seasonal variation in the deer population distribution, using a geographic automata model.

2. Materials and Methods

2.1. *Study site*

The study site, a 9 county area of southern Texas bordering Mexico (Figure 1), has been previously described [37, 96]. It consists of two Ecoregions, the Edwards Plateau (EP) in the north and the South Texas Brush (ST) in the south, which split the study region approximately in half (Figure 2). Seasonal variation in the overall study region is characterized by hot, dry summers and mild, moist winters, with average annual rainfall ranges between 750 and 1200 mm. Drought is common and periodically has effects on habitat resources and the wildlife population. The EP is home to the state's largest white tailed deer concentration and the population density in this Ecoregion is higher than in any other part of the state, with an estimated 100 deer per 405 hectares [88]. The ST Ecoregion is actively managed to support hunting for white tailed deer and population densities deer in this Ecoregion is considered moderate, with an estimated 29 deer per 405 hectares [88].

2.2 *Data source*

Bi-weekly composite NDVI images for 2006 (n=26) at 1 kilometer resolution were obtained for the study region from the USGS EROS Data Center. The baseline predicted distribution of deer in the study region was derived through Dasymetric mapping at the regional level as described by Highfield et al [37].

2.3 *Preparation of seasonal deer distributions*

A seasonal average NDVI coverage was derived and used to represent each of four seasons (winter, spring, summer and fall) for white tailed deer distributions. Seasonal-specific NDVI coverages were developed as follows. The 26 bi-weekly composite NDVI images were converted to rasters and projected for the study area. These 26 images were subsequently categorized into 4 seasons (December to February [winter], March to May [spring], June to August [summer] and September to November [fall]) and an average NDVI value at the pixel level for each of the seasons was calculated. Pixels in suitable habitats (as described in detail by Highfield et al. [36]) were extracted using a geographic information system (ArcGIS 9.1, ESRI Inc., Redlands CA). Briefly, pixels in shrub, forest and grass land use categories were extracted from the 1992 National Land Cover Dataset (NLCD) as suitable habitats and were used for creating seasonal distributions.

As a baseline, the white tailed deer population was assumed to be at equilibrium (number of births equal to number of deaths). The baseline predicted deer distribution

for this study, described in detail by Highfield et al. [37], was derived using disaggregation based on region (DR). Briefly, county-level deer populations were summed to a regional level and disaggregated based on suitable land use and estimated carrying capacity within each land use category derived from expert opinion [37]. Forest, shrub and grassland land use categories were extracted from the NLCD dataset. Estimated carrying capacity derived from expert opinion yielded values of 0.3 for forest and shrub and 0.1 for grassland for the study region. Based on the proportion of pixels within each land use category and the weight derived from expert opinion, the number of deer was proportionally distributed within each land use category. The resulting fractional counts of deer at a resolution of 30 meters were subsequently aggregated to a 1 km^2 integer grid [37].

To model the seasonal shift in the distribution of deer, a regression model was used. Two separate bivariate regression models, using NDVI as the independent variable and previously estimated deer density as the dependent variable, were estimated [3] and further diagnosed to quantify and correct for spatial dependence in the data. Prior to developing regression models the outcome (deer density) and predictor (NDVI) data were evaluated for a linear relationship using a correlation coefficient (Stata 10, Stata Corp., College Station, TX). Then an ordinary least squares (OLS) regression model was fit to the data for each season. The OLS residuals for each season were evaluated for the presence of significant ($P<0.05$) spatial autocorrelation using a global Moran's I statistic

[3]. Significant spatial autocorrelation violates the assumption of independent observations and can bias standard errors, increasing the likelihood of Type I errors.

Additional spatial diagnostics of the OLS models (robust Lagrange multipliers [LM]) were performed for each model if spatial autocorrelation was observed [3]. The inclusion of a spatial lag or spatial error term into an OLS regression equation can produce inconsistent results due to unaccounted for spatial autocorrelation and is considered inappropriate [4, 71]. Therefore, a spatial autoregressive model (spatial lag or error) using maximum likelihood estimation was selected when indicated by the LM tests. In cases where both the spatial lag and spatial error models were significant ($P<0.05$), based on the LM tests, both models were evaluated and the model producing the lowest log likelihood and highest pseudo R^2 statistic was selected. The selection of a lag distance for spatial autoregressive models can often be subjective. For this study, an assumed home range (2 km) for deer [13] was used to generate the weights matrix for the autoregressive lag models. Rho (ρ) is the coefficient of the spatial lag term (shown in Table V) and shows the spatial dependence inherent in the data by measuring the average influence on observations by their neighboring observations. The selected spatial autoregressive models for each season were evaluated for goodness of fit using a pseudo-R^2 statistic prior to simulating the FMD spread model. The residuals of the spatial autoregressive models were also graphically evaluated for normality. The predicted number of deer per pixel for each season was subsequently used as the data set in the simulation model.

2.4 *Simulation model*

The potential spread of FMD, by season and Ecoregion, within the deer population was simulated using a geographic automata model ("SIRCA") that has been previously described [21, 37, 96]. In the model deer could pass through four disease states: susceptible, latent, infected and immune. The probability of FMD virus transmission from one location to another was calculated as the product of the relative deer densities of the two locations, modified by the distance by which they were separated. Interactions were restricted to within a 2 km maximum neighborhood distance and up to a maximum of 8 neighbors [37, 96]. Locations containing more deer than a pre-specified maximum threshold value were assigned a probability of 1.0. The remaining locations were linearly scaled into the interval 0 to 1 by dividing each location's density by the maximum threshold value [37, 96].

To incorporate chance into the model, interactions between an infectious location and a susceptible neighbor occurred when a value from a pseudo-random number generator was below their joint probability threshold [37, 96]. Once a location was infectious the second, third, and fourth transitions in the model depend on the specified length of the latent, infectious and immune periods [37]. The specific values for each location were assigned randomly within the corresponding parameter ranges from a uniform distribution. As in previous studies, homogenous mixing was assumed to take place within but not between cells [37, 96].

The same baseline modeling scenario was used for all model comparisons: five cells (pixels) per Ecoregion (n=2) were randomly selected (SPSS 14.0, SPSS Inc., Chicago, IL) and selected as infected to initiate the simulation within each season (n=4). For every simulation of the model, each cell was allowed to interact with other cells within a 2 kilometer neighborhood (based on cell centroids) representing the home range of deer within the study area. The model was simulated for a time period representing 90 days to avoid overlap between seasons and 100 model runs were simulated for each dataset, yielding a total of 9,000 iterations for each season.

2.5 *Data analysis*

Descriptive statistics were calculated for each of the seasonal predicted distributions and the predicted spread of FMD in the population. The median number of deer infected and median area affected (km^2) were used to characterize each set of simulations at the 90^{th} model day. The predicted number of deer infected and the predicted number of infected locations (cells) for each model simulation (n=100) of each season (n=4) and Ecoregion (n=2) was evaluated for normality for further analysis. Due to extreme violations of normality, even after data transformations, a non-parametric Kruskal-Wallis (K-W) test was used to test for differences in predicted epidemic spread in the 8 treatments (Ecoregion and season). Miller's multiple comparison procedure was used to identify groupings in the predicted epidemic spread.

3. Results

Descriptive statistics for each seasonal deer distribution are shown in Table 16. A significant ($P<0.001$) linear relationship (correlation coefficients, 0.67, 0.6, 0.55, 0.59 for winter, spring, summer and fall, respectively) between deer density and NDVI was observed for all seasons. Significant ($P<0.001$) positive spatial autocorrelation was observed in the OLS regression residuals in each of the four seasons; Moran's I for winter, spring, summer and fall were 0.66, 0.71, 0.72, and 0.72, respectively. For all seasons, a spatial autoregressive lag model was preferred over a spatial autoregressive error model, based on log likelihood values. Characteristics of these fitted spatial autoregressive lag models are summarized in Table 17. Rho was >0.9 for all seasons, indicating that observations were heavily influenced by the surrounding values. Residuals from the spatial autoregressive lag model for each season visually appeared normally distributed. The spatial distributions predicted using the autoregressive lag models for each season are shown in Figure 17. Areas of high density deer distribution were more common in the winter season, although extensive areas of high deer density were predicted in the north-eastern parts of the study area in all seasons.

The predicted spread of FMD for each season and Ecoregion is summarized in Table 18 and shown graphically in Figures 18 through 21. Boxplots of the predicted spread of FMD for each season and Ecoregion are shown in Figure 22. There were always significantly higher numbers of infected deer in the EP Ecoregion, versus the ST

Ecoregion (Table 18). There were significant differences in epidemic spread by Ecoregion and season (Kruskal-Wallis chi-squared = 726.139, df = 7, p-value < 0.0001). The Miller's multiple comparison test indicated that within the EP Ecoregion, the highest number of infected locations (cells) occurred in winter, with the lowest in the spring and summer (tied by Miller's). The highest number of infected deer occurred in winter with the lowest in the summer. Within the ST Ecoregion, the highest number of infected locations (cells) and deer occurred in the fall and summer (tied by Miller's test), with the lowest in the winter.

4. Discussion

Substantial differences were observed in the median predicted magnitude of the FMD outbreak both by season and Ecoregion: the number of deer predicted to be infected ranged from 7,792 to 19,493. These differences can be explained by changes in modeled deer distribution within the study region, since all other parameters were held constant within this simulation study. Results suggest that the outcome of an FMD incursion in a population of wildlife, such as white tailed deer in south Texas, might depend on both where and during which time of year the incursion occurs.

The spatial autoregressive lag models using NDVI to predict deer distribution by season fitted the data well, as measured by the pseudo R^2 statistic (>0.8 for all seasons). We did not find substantial differences in the overall estimated number of deer in the study

region based on the spatial autoregressive lag model predicted distributions of deer (Table 16). However, the predicted spatial arrangement and continuity of the population varied substantially by season (Figure 17). Thus, the difference in predicted FMD spread within these populations is attributed to the spatial patterns present in the animal population – not the overall size of the population.

A significantly ($P<0.05$) higher number of predicted FMD infected deer and spatial locations (cells) were observed in the EP versus ST Ecoregion, regardless of season. Within Ecoregion, significant ($P<0.05$) differences in the predicted number of deer infected and number of spatial locations (cells) infected was observed by season. Winter in the EP Ecoregion resulted in both the highest number of infected deer and locations (cells), whereas summer and fall resulted in the highest number of infected deer and locations (cells) in the ST Ecoregion. These results further support previous work [37] which showed that the spatial continuity of a population plays a major role in the predicted outbreak size. As previously reported, this result is not surprising given that the SIRCA model is a local-based spatial disease spread model [37]. The more continuity in the spatial distribution, the better the opportunity for interactions between locations and therefore more interactions occur, even when there are lower interaction probabilities [37]. Thus, our observations are consistent with epidemic theory and the importance of spatial heterogeneity [45, 48].

The model used in this study has been used previously to investigate wildlife-domestic species interactions between feral pigs and cattle [21, 96], between wild deer and cattle [96] and to evaluate the impact of spatial estimation methodologies on model predicted spread of FMD in deer [37]. In the current study, the focus was on extending previous work to incorporate seasonal variability in white tailed deer population and to subsequently predict how the spread of FMD may vary in the study region by season. As in a previous study [37], it was assumed that due to low grazing densities of cattle in this extensively managed livestock system, cattle populations did not contribute greatly to disease spread. This study focused on the initial stages of disease spread (≤90 days) in order to assess seasonal variability [37]. It was assumed that the average home range of deer (2 km) was adequate for creating spatial weights for the spatial autoregressive lag models. Given that deer show high fidelity to their home range, this assumption is likely to be valid [51]. However the spatial scale of the influence of the surrounding population on seasonal deer distribution is unknown. Future work should incorporate varied spatial weights and assess how this variation might impact model predictions of deer distribution.

Behavior of wildlife species, such as deer, will also vary by season and should be included in future work focusing on the spread of FMD in wildlife populations over time. For example, the rut (breeding season) in white tailed deer in the study area typically occurs in the EP Ecoregion between October and December and in the ST Ecoregion in December [87]. During this time of the year, bucks are more likely to move

around their environment and cover larger distances than normal [87]. This could contribute substantially to increased spread of FMD due to a greater numbers of interactions with other potentially susceptible deer. Juvenile males will also disperse from their female groups and an increase in the number of single males in the population may need to be modeled [63]. In addition, a stable population (no birth or death) was assumed for this study. Future studies should incorporate changes in the population due to births and deaths, especially given that this area is intensively managed for hunting and recreation.

An assumption was also made that the same spatial relationship for predicting deer distribution (in the autoregressive lag models) was valid over the entire study area (both Ecoregions). Ecoregions are broad ecological zones comprised of similar soils, topography, land use and vegetation (habitat). Given the substantial differences in the spatial distribution of deer in the 2 Ecoregions in the study area, it is likely that variation in the spatial relationship may exist between Ecoregions. Future work should examine the application of regression models specific to Ecoregion, to determine if there is variation. There are likely differences in the spatial distribution of deer by Ecoregion and there is utility in modeling separate regression models by Ecoregion. However, the usefulness of Ecoregion as a predictor for estimating deer distribution might be limited, since habitat variability is captured at a finer resolution by using land use data. Using Ecoregions as a marker for modeling deer behavior is also limited because regions are a very large scale measurement of the environment and have no associated attribute data.

While it might be useful to model deer behavior with a larger number of finer resolution ecological zones, it becomes exceedingly complex as data requirements increase, a greater number of variables have to be estimated and information on behavior within a particular ecological area has to be derived from expert opinion. This greatly adds to uncertainty in the resulting estimates.

The NDVI as a measure of forage availability and the relationship to deer distribution has been evaluated in previous studies [59, 83]. For this study, a single year of NDVI data was used and bi-weekly measurements were grouped into a seasonal average to predict deer distribution. As documented in previous studies [59, 83], a traditional seasonal (winter, spring, summer, fall) breakdown was assumed to be appropriate. More detailed analysis of methods of grouping NDVI data for predicting deer distribution is warranted, as a traditional season approach may not adequately capture seasonal variability in the relationship between vegetative greenness and forage availability. It was further assumed that one year of NDVI data was adequate to model seasonal variability. This assumption is valid if the interest in modeling deer distribution is focused on the most recent year; however, longer term trends may also be of interest to modelers and policy decision-makers. Future work on a short time series might provide a better understanding of the broad patterns of NDVI over time in the study area.

Based on a review of the literature, this is probably the first study to incorporate seasonal variability in wildlife distributions and to define the potential magnitude of an FMD

outbreak by season. High levels of seasonal variability in the model predicted spread of FMD were found. Future work focusing on improved methods of analysis of NDVI data, spatial regression models and incorporating behavioral traits are needed to yield additional insights into the potential spread of foreign animal diseases in wildlife populations.

CHAPTER V

THE IMPACT OF POLICY MANDATED MITIGATION STRATEGIES ON THE PREDICTED SPREAD OF FOOT AND MOUTH DISEASE IN WHITE TAILED DEER IN SOUTH TEXAS

Modeling the potential spread of disease in wildlife populations is an important tool for predicting, responding to and recovering from a foreign animal disease incursion. The potential role of policy mandated mitigation strategies on the spread of foot and mouth disease (FMD) in wildlife populations has not been evaluated. We conducted a series of simulation experiments to determine how pre-emptive mitigation strategies applied to white tailed deer populations might impact the predicted magnitude and distribution of foot and mouth disease outbreaks in south Texas. Outbreaks were simulated using a susceptible-infected-recovered geographic automata model ("SIRCA"). The study region was a 9-county area (24,000 km^2) of southern Texas, comprising 2 distinct Ecoregions. A previously derived seasonal deer distribution was used to represent the time of year with the highest and lowest spatial continuity in the 2 Ecoregions, respectively. The magnitude of the predicted outbreaks for each mitigation and Ecoregion were evaluated by comparing the median number of deer infected and median number of spatial locations infected, respectively, across 4 mitigations and 2 Ecoregions. A non-parametric Kruskal-Wallis test was used to test for differences in predicted epidemic spread in the 8

treatments (Ecoregion and mitigation). Miller's multiple comparison procedure was used to look for groupings in the predicted epidemic spread. Substantial differences were observed in the median predicted magnitude of the FMD outbreak both by mitigation and Ecoregion: the number of deer predicted to be infected ranged from 1,054 to 4,858. Results suggest that the outcome of an FMD incursion in a population of wildlife, such as white tailed deer in south Texas, might depend on both where the incursion occurs and the type of pre-emptive mitigation strategy applied, if any.

1. Introduction

The aim of this research is 1) To review the existing foot and mouth disease FMD response policies for wildlife and, 2) To investigate, using a simulation model, the potential impact of various mitigation strategies on the predicted spread of FMD in white tailed deer in south Texas. This chapter will provide critical insight into the current response policy for FMD incursions in wildlife populations in the United States and allow for a quantitative evaluation of the potential spread of FMD in white tailed deer, given the effect of various mitigation strategies.

The *Office International des Epizootes* (OIE, World Animal Health Organization) is the international governing body which sets rules for international trading and assigns trading status to countries with respect to importation and exportation of animals and animal products. The OIE is responsible for certifying freedom from disease for FMD. If

FMD were to be detected in a country (such as the United States) currently certified as free from the disease, the OIE would remove the country's trade eligibility for a period of not less than 6 months following the date of successful disease eradication. If the United States were to vaccinate to control the disease, the OIE would not reinstate the trade status until at least 12 months after the last vaccinated animal was slaughtered [90]. Under the current regulations, there is no distinction made between vaccinated livestock and vaccinated wildlife [90].

The United States Department of Agriculture (USDA), Animal and Plant Health and Inspection Service (APHIS) is the government agency responsible for responding to and controlling foreign animal disease incursions in the U.S. The response policies for foreign animal disease incursions are published in the National Animal Health Emergency Management System Guidelines (NAHEMS) [69]. The following section of this chapter summarizes the NAHEMS guidelines.

Within the APHIS, the Wildlife Section is responsible for handling foreign animal disease incursions that involve a wildlife species. The Wildlife Section includes a Wildlife Coordinator, Wildlife Officers, a State Wildlife Liaison and necessarily field personnel such as wildlife biologists. Field personnel are selected, at the discretion of the wildlife officer, either from federal or state wildlife related agencies. Field personnel are expected to supply all necessary field equipment until the federal government is able to provide additional resources (if necessary). The current guidelines establish infected and

surveillance zones around infected livestock premises, and establish the size of the infected and surveillance zones to be 10km and 20km, respectively. In the initial assessment of the outbreak, the risk of wildlife (all free-ranging native, feral and exotic animals) involvement will be assessed as follows: wildlife species present in the area, susceptibility and infectiousness of the species, level of exposure to domestic animals and the disease agent.

If a risk of wildlife involvement is suspected, surveillance of wildlife in the area surrounding infection is mandated. Both active and passive surveillance methods will be used. In the case of FMD, active surveillance will entail lethal collection of samples from surrounding ruminants (deer) and feral hogs for diagnostic testing, carcass searches and road-kill investigation. Passive surveillance will include morbidity and mortality surveillance based on reports received by the Wildlife Section. If wildlife were identified as a risk factor for disease spread or persistence, local reduction of the density of the population in the infected zone would be conducted. In addition, efforts to reduce contact between wildlife and domestic species may be used. Options would include the creation of barriers including: a depopulation buffer, a vaccination buffer, fencing, habitat alteration and hazing. Long term surveillance would be required to ensure that the disease has been eradicated. While FMD has not been present in the United States since 1929 [47], other diseases which also have a wildlife component are found in the U.S. and may provide insight into potential mitigation strategies.

One example of a disease with a wildlife component in recent management history is bovine tuberculosis (TB) in Michigan. Bovine TB was first found in a single white tailed deer in northeastern Michigan in 1975 [36]. Believed to be an isolated case, it was not until 1994 that another infected deer was found and surveillance began. Currently the disease prevalence in the deer population in the infected area is approximately 3% [36]. Cattle in proximity to the deer were tested in 1995 and found negative, indicating that the infection was maintained within the deer population [36]. It is believed that the deer in the area were initially infected by cattle. Due to management practices (such as supplemental feeding), the disease has since been maintained in the white tailed deer population [36]. In recent years, on average 2.2 cattle herds per year have been infected from contact with white tailed deer [36]. Analysis of available data has found a significant positive relationship between supplemental feeding of white tailed deer, deer density and the prevalence of the disease. Correlations of 0.7 were observed for both supplemental feeding and deer density individually. Regression analysis indicated that both factors accounted for 55% of the variation in the prevalence within the infected area [36].

Another example of a disease with a wildlife component is brucellosis. The primary mitigation strategy for brucellosis is to reduce contact between wildlife and livestock in the Yellowstone National Park (YNP) [66]. To achieve this goal, a Northern and Western boundary has been established around YNP. Each of the boundary areas has three management/surveillance zones, where various mitigation strategies are applied.

Hazing is used to discourage bison from leaving YNP, and any that do not respond are captured and tested for brucellosis. Seropositive animals are sent to slaughter; the first 100 that test negative are released, with the remaining going to slaughter. Pregnant Bison that test negative are permitted to leave only after cattle have been removed (fall and winter). If cattle are kept on private lands during the fall and winter, a buffer zone (Bison free) is maintained until after the cattle are removed. Cattle in the area surrounding YNP are required to be vaccinated.

Additional issues that may influence disease eradication and control include carcass disposal and hunting. Carcass disposal for wildlife follows similar regulations to that in domestic species. Carcasses may be disposed of onsite or transported to a central location within the infected or surveillance zone designated for disposal. Hunting activities would be banned within the infected and surveillance zones; however, hunting might be banned at a larger regional scale, depending on the situation.

The last outbreak of FMD in the United States involving wildlife occurred in California in 1929 [47]. Depopulation was the selected method of disease control and more than 22,000 deer were slaughtered over a 2 year period to eradicate the disease [47]. If the disease were to occur in the United States today, the role of wildlife in the potential spread and maintenance of the disease is unknown. There are an estimated 406,000 white tailed deer in the study area, an area comprising of approximately 24,500 square km and 9 counties in south Texas (Figure 1). White tailed deer represent an important

financial resource to a substantial number of ranchers in south Texas [14], and the deer population is actively managed for hunting and recreation purposes [14, 89]. White tailed deer hunting has a large economic impact in the area [14, 89]. This encourages many ranchers to supply supplemental feed and manage their properties to promote the presence of white tailed deer, in addition to cattle [14, 89, 97]. Given the length of time since FMD last occurred in the United States and the unknown role that wildlife might play in an outbreak in an extensive livestock management system such as south Texas, disease modeling provides an important tool for evaluating the potential impact of the disease and mitigation strategies prior to an actual outbreak. Modeling may also be used during an outbreak to inform response strategies, particularly for wildlife populations. Modeling predictions may also be used to guide policy prior to an outbreak.

The objectives of this study were: 1) To characterize the current response policy for FMD in wildlife populations in the United States; 2) Describe the predicted FMD outbreak that might be observed within a white tailed deer population, using the geographic automata model; and 3) Evaluate the impact of various pre-emptive mitigation strategies and their effectiveness for controlling predicted FMD spread within a white tailed deer population.

2. Materials and Methods

2.1. *Study site*

The study site, a 9-county area of southern Texas bordering Mexico (Figure 1), has been previously described [37, 96]. It consists of two Ecoregions, the Edwards Plateau (EP) in the north and the South Texas Brush (ST) in the south (Figure 2). Seasonal variation in the overall study region is characterized by hot, dry summers and mild, moist winters, with average annual rainfall ranging between 750 and 1200 mm. The EP Ecoregion is predominately rangeland and is home to the highest concentration of deer in Texas. The ST Ecoregion is considered a brush community and is home to a moderate density of white tailed deer. White tailed deer hunting has increased in this Ecoregion and the vegetation is actively managed to support hunting [88].

2.2 *Data source*

Estimated distributions of deer in the study region during the winter season (Chapter III of this dissertation) were used to represent the deer population. Winter was selected because it is the season with the highest (EP Ecoregion) and lowest (ST Ecoregion) spatial contiguity in the predicted distribution of deer. Using winter therefore allows for a “worst” and “best” case representation of the potential spread of FMD in the population. Winter deer distributions was derived using a spatial autoregressive lag model and the Normalized Vegetation Difference Index (NDVI) to predict the seasonal shift in deer distribution in the study region (Chapter IV of this dissertation).

2.3 *Simulation model*

The spread of FMD in the white tailed deer population in the study region was examined using a geographic automata model ("SIRCA") previously described [21, 37, 96]. In the model, deer can pass through four disease states: susceptible, latent, infected and immune. The probability of FMD virus transmission from one location to another was calculated as the product of the relative densities of the two locations, modified by the distance by which they were separated. Interactions were restricted to within a 2 km maximum neighborhood distance and up to a maximum of 8 neighbors [37, 96]. Locations containing more deer than a pre-specified maximum threshold value were assigned a probability of disease transmission of 1.0. The remaining locations were linearly scaled into the interval 0 to 1 by dividing each location's density by the maximum threshold value [37, 96].

To incorporate chance into the model, interactions between an infectious location and a susceptible neighbor occurred when a value from a pseudo-random number generator was below their joint probability threshold [37, 96]. Once a location was infectious the second, third, and fourth transitions in the model depend on the specified length of the latent, infectious and immune periods [96]. The specific values for each location were assigned randomly within the corresponding parameter ranges from a uniform distribution. As in previous studies, homogenous mixing was assumed to take place within but not between cells [37, 96].

Three pre-emptive mitigation strategies were evaluated for each of the two Ecoregions (EP and ST). In addition, a baseline scenario was simulated, in which no mitigation strategies were applied. Thus, a total of eight scenarios were simulated. All spatial processing of the pre-emptive mitigation strategies was done using a geographic information system (ArcGIS 9.1, ESRI Inc., Redlands CA). All mitigation strategies were applied prior to simulating the spread of FMD in the Geographic Automata ("Sirca") model.

The first mitigation strategy evaluated was a pre-emptive targeted cull. Deer locations with a density of 20 or more were identified and targeted for depopulation. These locations were removed from the input dataset by setting the density to zero prior to simulating an FMD virus incursion in the study region.

The second mitigation strategy evaluated was a cull of deer at locations selected at random. This strategy might represent the impact of allowing hunters to remove deer from the study area. Ten percent of the overall population was randomly selected by choosing 3,059 locations within the study region (SPSS 14, Chicago, IL). These locations were then culled (density set to zero) to evaluate the impact of an overall pre-emptive cull of the population. Locations with a higher density of deer were given preference for depopulation, assuming that hunters would be more likely to have "success" in these higher density areas. Locations with >15 deer comprised 70% of the 3,059 locations selected for culling.

The third mitigation strategy evaluated was a targeted depopulation buffer (DB). Deer locations that were within a 1 km distance of cattle herds, that contained a population of 50 or more head, were depopulated to create a buffer surrounding at-risk cattle herds. Cattle premise locations were obtained from the U.S. Department of Agriculture, Farm Services Agency (FSA).

For each scenario within the 3 mitigation strategies, as well as the baseline scenario, 1 cell was randomly selected within each of the two Ecoregions. Prior to simulation, each of these locations was set to infected status to initiate the model. Thus, 1 initiation site was selected for the 4 mitigations (baseline, targeted cull, random cull, depopulation buffer) within each Ecoregion. For every simulation of the model, each cell within the study region was allowed to interact with other cells within a 2 kilometer neighborhood (based on cell centroids), representing the assumed home range of deer within the study region. For each of the scenarios, the model was simulated for a time period representing 100 days and 100 model runs were simulated for each, yielding a total of 10,000 iterations for each of the 8 scenarios simulated.

2.4 *Data analysis*

Descriptive statistics were calculated for the predicted number of deer in the study region (pre- and post-mitigation) and the predicted spread of FMD in the population for the baseline (no mitigation applied) and each of the 3 pre-emptive mitigation strategies.

The median number of deer infected and median area affected (km^2) were used to characterize each set of simulations at the 100^{th} model day. The predicted number of deer infected and the predicted number of infected locations (cells) for each model simulation (n=100) of each mitigation (n=4) and Ecoregion (n=2) was evaluated for normality for further analysis. Due to extreme violations of normality, even after data transformations, a non-parametric Kruskal-Wallis (K-W) test was used to test for differences in predicted epidemic spread in the 8 treatments (Ecoregion x Mitigation). Miller's multiple comparison procedure was used to identify groupings in the predicted epidemic spread.

3. Results

Descriptive statistics for each mitigation strategy are shown in Table 19. The mitigation strategies resulted in large differences in the predicted population size (range 178,373 – 406,667) and spatial contiguity of the population (Figure 23). The predicted spread of FMD for each Ecoregion and mitigation strategy is summarized in Table 20 and shown in Figures 24 through 27. There were significantly higher numbers of infected deer predicted in the EP Ecoregion versus the ST Ecoregion for all mitigations except the targeted depopulation strategy (Table 20). There were significant differences in epidemic spread by Ecoregion and mitigation strategy (Kruskal-Wallis chi-squared = 698.018, df = 7, p-value < 0.0001). The Miller's multiple comparison test indicated that within the EP Ecoregion, the highest number of infected locations (area, km^2) occurred with no

mitigation (baseline scenario), and the lowest number of infected locations occurred with the depopulation buffer mitigation strategy. The second lowest number of infected locations resulted from the targeted depopulation strategy, whereas random depopulation was ranked third. The highest total number of infected deer occurred with both the baseline (no mitigation applied) and random depopulation strategies, whereas the depopulation buffer (DB) strategy resulted in the lowest total number of infected deer. Targeted depopulation resulted in the second lowest total number of deer infected. Within the ST Ecoregion, the highest number of infected locations (area, km^2) were observed with both the baseline and targeted depopulation mitigation strategies (tied by Miller's test). The depopulation buffer (DB) mitigation strategy produced the lowest number of infected locations. Random depopulation resulted in the second lowest number of infected locations. The highest total number of infected deer in the ST Ecoregion was observed with the baseline, targeted and random depopulation mitigations (tied by Miller's test). Only the depopulation buffer (DB) mitigation strategy resulted in a lower total number of infected deer.

4. Discussion

Substantial differences were observed in the median predicted magnitude of the FMD outbreak both by Ecoregion and mitigation: the number of deer predicted to be infected ranged from 1,054 to 4,858. These differences can be explained by differences in the

mitigation strategies simulated within the study region, via the effect on deer distributions. Results suggest that the outcome of an FMD incursion in a population of wildlife, such as white tailed deer in south Texas, might depend on both where the incursion occurs (the type of landscape, represented by Ecoregion in this study), and the type of mitigation strategy (targeted cull, random cull and depopulation buffer simulated in this study) that might be applied.

In both Ecoregions studied, the depopulation buffer (BD) mitigation strategy produced the lowest number of both infected locations (area infected, or the size of the "infected zone") and the total number of deer infected. This mitigation drastically reduced both the overall population size and spatial contiguity (Table 19, Figures 23 through 27), which explains the smaller outbreaks that were observed. Previous research has shown that spatial contiguity has an important role in the resulting predicted spread of FMD in the Geographic Automata ("SIRCA") model [37].

In this study, we used a 1 km depopulation buffer surrounding "at risk" livestock herds because our interest was in simulating disease spread and mitigation strategies in the white tailed deer population only. However, the current regulations state only that a "local reduction" of wildlife in the infected zone (10 km surrounding each infected livestock herd) would be used. There is no mention of pre-emptive mitigation approaches in the current regulations. They are also unclear regarding how the local reduction policy would be implemented. It is unknown if a 10 km buffer around each

infected livestock herd would be necessary or the most efficient approach for controlling the spread of the disease. Future studies simulating infected livestock locations with depopulation of surrounding wildlife could give policy decision-makers insight into the most effective buffer size and the risks and benefits of pre-emptive versus post disease detection mitigation strategies for areas involving high densities of wildlife.

The effects of the other mitigation strategies simulated were not consistent across the two Ecoregions studied. In the EP Ecoregion, the targeted cull mitigation produced the second lowest number of both locations and deer infected. Given the high density of deer in the EP Ecoregion, it is not surprising that targeted depopulation of high density cells (>20 deer) resulted in significantly reduced spread of FMD.

Random depopulation, used to simulate the effect of hunters removing deer, had little impact on the predicted spread of FMD through deer populations in either of the Ecoregions. In the EP Ecoregion, random depopulation was equivalent to the baseline (no mitigation) strategy. In the ST Ecoregion, random depopulation reduced the number of locations infected, but not the number of deer infected. These findings indicate that more than 10% of the overall population would need to be removed in order to have a substantial impact on reducing the likely spread of FMD. To estimate the actual proportion of the population that would need to be removed, additional studies should incorporate a sensitivity analysis approach and a range of culling percentages. However, even removing 10% of the population is likely to be logistically difficult and costly.

The only mitigation strategy that reduced the number of deer infected in the ST Ecoregion was the depopulation buffer (DB). The differences observed in mitigation strategy by Ecoregion indicate that different mitigation strategies might be preferred, depending on the composition of the deer population at-risk for infection. Thus, the policy for responding to an FMD virus incursion that involves an uncontrolled animal population, such as white tailed deer in southern Texas, needs to be tailored to the ecological region of concern. For example, it is unlikely that one, simple policy would be successful in all regions of the United States, or even within Texas.

The Geographic Automata ("SIRCA") model has been used previously to investigate wildlife-domestic species interactions between feral pigs and cattle [21, 96], between wild deer and cattle [96], to evaluate the impact of spatial estimation methodologies on model predicted spread of FMD in deer [37] and to evaluate differences in predicted spread of FMD in deer by season [Chapter III of this dissertation]. In the current study, the focus was on extending previous work to incorporate pre-emptive mitigation strategies that might be considered to minimize the risk of white tailed deer becoming reservoirs of FMD virus infection, should an incursion occur. This study does not directly address the situation of dealing with an outbreak of FMD in white tailed deer, once it has already begun. Although one or more of the strategies simulated might play a role in such a situation, additional strategies might need to be considered. The use of buffers to contain FMD in wild animal populations has been successful elsewhere [10],

and research on the use of buffers in south Texas in the case of an FMD virus incursion should be considered.

As in our previous studies [37], we made the assumption that due to low grazing densities of cattle in this extensively managed livestock system, cattle populations did not contribute greatly to disease spread. This assumption could be tested by extending the model to include such dynamics. We also focused on the initial stages of disease spread (≤100 days) in order to assess the effectiveness of the mitigation strategies [21, 96]. It is likely that if FMD was not controlled after a period of 100 days, disease eradication in the short term may no longer be the goal. In such circumstances, a more complex model might be needed to investigate the best way of responding to the disease.

While some of the pre-emptive mitigation strategies simulated in this study reduced the predicted spread of FMD in white tailed deer in the study region, there may be political and practical issues with these approaches. Deer are an important financial and recreational resource in the study area, and many other regions of the U.S. [14, 89]. Widespread public opposition to pre-emptive depopulation may be a political barrier to implementing these strategies. Further, the resources required to depopulate the wildlife population would likely reduce available resources for responding to FMD virus infection in livestock populations in the study region, if it occurred. Given resource constraints, it may not be practical to implement all of the simulated strategies, especially those requiring depopulation of large numbers of deer. Future studies

incorporating the potential role of barriers, such as deer proof fencing, are necessary to gain further insight into potential mitigation strategies and their effect on the predicted spread of FMD in wildlife. Deer proof fencing may offer the same benefits in reducing disease spread as the depopulation buffer, by reducing the ability for deer to come into contact with each other and livestock, but offer the added benefit of not requiring substantial depopulation of the deer population. In addition, if deer are at-risk of becoming a potential reservoir for the disease or if it is impractical or impossible to completely eradicate the disease in wildlife, it may be possible to create FMD endemic zones using a barrier approach similar to the approach used in Kruger National Park in South Africa [10]. It might be possible to also incorporate landscape features, such as rivers, mountains, or major roads, to increase the effectiveness of a buffer zone. However, planning prior to the incursion of a foreign animal disease is needed, so that policies can be in place and an appropriate buffer can be identified in a timely manner. Further research in this area is warranted.

This is the first study to define the potential magnitude of an FMD outbreak in a wildlife population, incorporating potential mitigation strategies. High levels of variability in the model predicted spread of FMD based on the mitigation strategy employed were found. Future work, focusing on the potential role of additional mitigation strategies, such as barriers, are needed to yield additional insights into the potential spread and mitigation of foreign animal diseases in wildlife populations.

CHAPTER VI

CONCLUSION

FMD is a highly contagious, transboundary disease of cloven-hoof animals and has long been considered the most dangerous foreign animal disease that might be inadvertently introduced into the United States [22]. FMD control strategies, mostly directed at livestock, seek to minimize the economic costs associated with loss of trade. The potential role of wildlife species, which may serve as disease reservoirs, has been largely overlooked. The presence of non-domesticated reservoir animal species is a serious barrier to effective control of FMD outbreaks [74, 85].

The United States has been free of FMD since 1929, following a number of outbreaks in California and Texas during the 1920's. The 1924 Californian outbreak involved deer which were exposed via contact from cattle [47]. Since the disease has not been present in this country for such a lengthy period of time, the entire population of cloven-hoofed animals in the United States is susceptible to infection. This includes livestock and wildlife species. Epidemic models represent an important tool to aid decision-making and epidemic response to foreign animal disease incursions. In the face of an outbreak, it is crucial that appropriate control measures be applied rapidly to control the disease. However, in most cases decisions regarding mitigation strategies must be made with

little current or empirical data and in the context of political, economic and social pressures. Disease spread models can give guidance on the probable extent and time span of an outbreak. They can also be used to evaluate the design of optimal control strategies, for policy formulation, for gap analysis and to develop and refine research agendas when disease is not present. However, for disease spread models to be used to their full potential it is critical that links between modelers and policy decision-makers exist *a priori*.

The conditions under which wild and feral animal species might become reservoirs of FMD virus, following an incursion into a country free of disease, are unknown. However, several factors – including population density and distribution, habitat requirements, social organization, age structure, home range, and barriers to dispersal – are likely to be important. This has been shown with bovine tuberculosis in Michigan, where deer density was found to be significantly correlated with increased prevalence of disease [36]. Because of the paucity of information and experience with FMD in reservoir species, simulation modeling is perhaps the only option for exploring the impact of an FMD virus incursion and therefore developing response plans and formulating policy [86]. With that in mind, this research project evaluated the potential role of wildlife, specifically white tailed deer, in the spread of FMD in an extensive livestock management system in Texas.

In the second chapter of this dissertation, the potential effects of geostatistical methods for estimating white tailed deer distributions were evaluated. Substantial differences in the estimated number of deer in the study region, based on the geostatistical estimation procedure used, were found: the total deer population ranged from 385,939 to 768,493.

Substantial differences were also observed in the median predicted magnitude of the outbreak, which ranged from 1,563 to 8,896 deer infected. This variability in the predicted median outbreak size, as a result of using different geostatistical methods to describe the population at-risk, supports the argument that reporting only summary statistics from simulation models can be misleading. It is important that an attempt be made to consider the entire predicted outbreak distribution when summarizing modeling results, especially if these results are to be presented to policy-makers or to be used by decision-makers in the face of an outbreak. Thus, the choice of geostatistical method for representing animal species distribution is probably secondary to the objectives of the study. If the aim is to estimate the overall impact of an FMD disease outbreak, results from this study suggest that the choice of geostatistical method is not critical. However, from the perspective of spatial analysis and predicting the likely spatial distribution of infected areas, the choice of geostatistical method becomes more important.

In the third chapter of this dissertation, the potential effects of critical model parameters on predicted FMD spread in wildlife reservoirs was evaluated. Substantial differences in the estimated number of deer infected and number of locations infected for various

parameter values were found: the total number of deer infected ranged from 3,772 to 119,873, while the total number of locations infected ranged from 227 to 6,526.

This variability in the predicted median outbreak size, as a result of using different parameter values, indicates that the model is sensitive to the input data. In particular, the model appears to be most sensitive to the length of the latent period and the number of neighbors (contacts), compared to a baseline scenario. It is important that an attempt be made to obtain the best available data when developing a disease spread model. Whilst every effort has been made in this dissertation to incorporate the best available estimates for a range of disease spread parameters, these parameters still have associated uncertainties and it is therefore important to understand how such uncertainty impacts model predictions.

The need to use spatially-explicit models to simulate the spread of FMD has been recognized [33 45], and incorporating spatial heterogeneity has been identified as possibly the greatest challenge to realistically representing FMD spread through a landscape [22]. In addition to capturing the spatial heterogeneity of the population across the landscape, wildlife distributions need to be seasonally-dynamic, since these species are particularly affected by variations in climate and natural resources.

In the fourth chapter, the potential role of seasonal variability in white tailed deer populations on the model predicted spread of FMD was evaluated. Substantial

differences were observed in the median predicted magnitude of the FMD outbreak both by season and Ecoregion: the number of deer predicted to be infected ranged from 7,792 to 19,493. These differences can be explained by changes in modeled deer distribution within the study region, since all other parameters were held constant within this simulation study. Results suggest that the outcome of an FMD incursion in a population of wildlife, such as white tailed deer in south Texas, might depend on both where and during which time of the year the incursion occurs. Within Ecoregion, significant differences in the predicted number of deer infected and number of spatial locations (cells) infected was observed by season. This is consistent with previous research [21] which found differences in the model predicted spread of FMD by season in feral hogs in Australia.

In the fifth chapter, the role of potential mitigation strategies on the model predicted spread of FMD was evaluated. Substantial differences were observed in the median predicted magnitude of the FMD outbreak both by Ecoregion and mitigation: the number of deer predicted to be infected ranged from 1,054 to 4,858. These differences can be explained by differences in the mitigation strategies simulated within the study region, via the effect on deer distributions. Results suggest that the outcome of an FMD incursion in a population of wildlife, such as white tailed deer in south Texas, might depend on both where the incursion occurs (the type of landscape, represented by Ecoregion in this study), and the type of mitigation strategy (targeted cull, random cull and depopulation buffer simulated in this study) that might be applied.

In both Ecoregions studied, the depopulation buffer (BD) mitigation strategy resulted in the lowest number of both infected locations (area infected, or the size of the "infected zone") and the total number of deer infected. This mitigation drastically reduced both the overall population size and spatial contiguity (Table VII, Figures II and III), which explains the smaller outbreaks that were observed.

While some of the pre-emptive mitigation strategies simulated in this study reduced the predicted spread of FMD in white tailed deer in the study region, there may be political and practical issues associated with these approaches. Deer are an important financial and recreational resource in the study area, and many other regions of the United States [14, 89]. Widespread public opposition to pre-emptive depopulation may be a political barrier to implementing these strategies. Furthermore, the resources required to depopulate the wildlife population would likely reduce available resources for responding to FMD virus infection in livestock populations in the study region, if it occurred. Given resource constraints, it may not be practical to implement all of the strategies simulated, especially those requiring depopulation of large numbers of deer.

This research project has allowed for an initial assessment of the potential spread of FMD in wildlife populations. However, more research is needed. Better estimates of critical model parameters are needed, and future research focused on collecting data that allows estimation of these parameters is required. Future work on the role of seasonal variability in model predicted spread should incorporate varied spatial weights and

assess how this variation might impact model predictions of deer distribution. Behavior of wildlife species, such as deer, will also vary by season and should be included in future work focusing on the spread of FMD in wildlife populations over time. For example, the rut (breeding season) in white tailed deer in the study area typically occurs in the EP Ecoregion between October and December and in the ST Ecoregion in December [87]. During this time of the year, bucks are more likely to move around in their environment and cover larger distances than normal [87]. This behavioral phenomenon could contribute substantially to increased spread of FMD because of increased numbers of contacts with other potentially susceptible deer. Juvenile males will also disperse from their female groups and an increase in the number of single males in the population may need to be modeled [63].

Future work focusing on improved methods of analysis of NDVI data, spatial regression models and incorporating behavioral traits are needed to yield additional insights into the potential spread of foreign animal diseases in wildlife populations. Future studies incorporating the potential role of barriers, such as deer-proof fencing, are also necessary to gain additional insight into potential mitigation strategies and their effect on the predicted spread of FMD in wildlife. Deer-proof fencing may offer the same benefits in reducing disease spread as the depopulation buffer, by reducing the ability for deer to come into contact with each other and livestock, but offer the added benefit of not requiring a substantial depopulation of the deer population. In addition, if deer are at-risk of becoming a potential reservoir for the disease, or if it is impractical or impossible to

completely eradicate the disease in wildlife, it may be possible to create FMD endemic zones using a barrier approach similar to the approach used in Kruger National Park in South Africa [10]. It might be possible to also incorporate landscape features, such as rivers, mountains, or major roads, to increase the effectiveness of a buffer zone. However, planning prior to the incursion of a foreign animal disease is needed, so that policies can be in place and an appropriate buffer can be identified in a timely manner. Future work, focusing on the potential role of additional mitigation strategies, such as barriers, are needed to yield additional insights into the potential spread and mitigation of foreign animal diseases in wildlife populations.

Texas is the largest cattle production state in the United States and offers the unique opportunity to develop, validate and model the potential impact of foreign animal diseases, such as FMD, in the U.S. agricultural industry. This study allowed the opportunity for an assessment of the impact of disease in one of the most critical locations of cattle production in the country. In general, models developed in Texas to predict areas at-risk of FMD from wildlife reservoirs should be applicable to other similar cattle producing areas of the United States where wildlife reservoirs are present. The decision-support system developed in the studies described in this dissertation provide decision-makers and those designing and implementing disease response and control policy with information on the potential spread of a foreign animal disease incursion with a likely wildlife reservoir. Use of such a decision-support system would enhance the disease incursion preparedness and response capacity of the United States.

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APPENDIX A

MODEL PARAMETERS

Parameter	Value
Latency, days (min, max)	3 – 5
Duration of infectiousness, days (min, max)	3 – 14
Duration of resistance to re-infection, days (min, max)	90 – 180
Maximum number of neighboring cells with which each infected cell can interact	8
Maximum distance of neighboring cells within which each infected cell can interact (meters)	2000
Density scaling parameters (min, max)	0 – 30

APPENDIX B

FIGURES

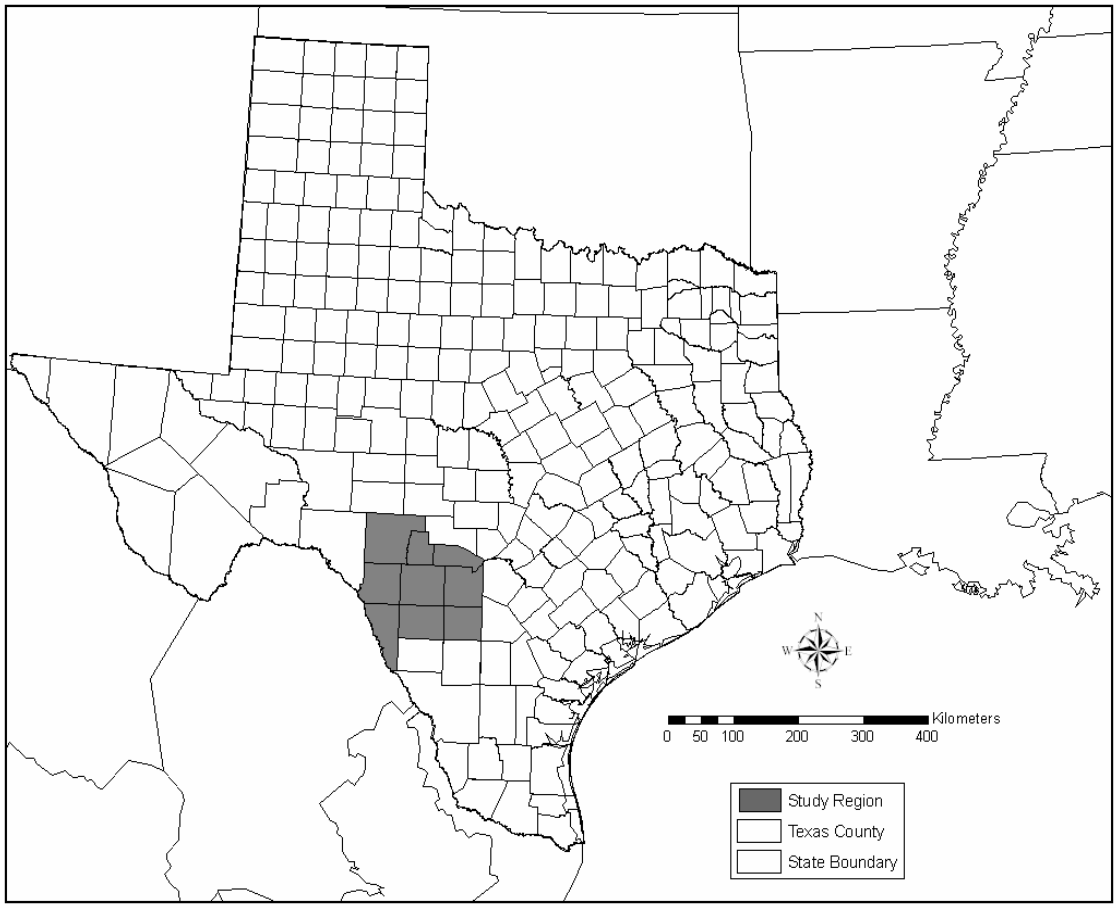

Figure 1. South Texas study region.

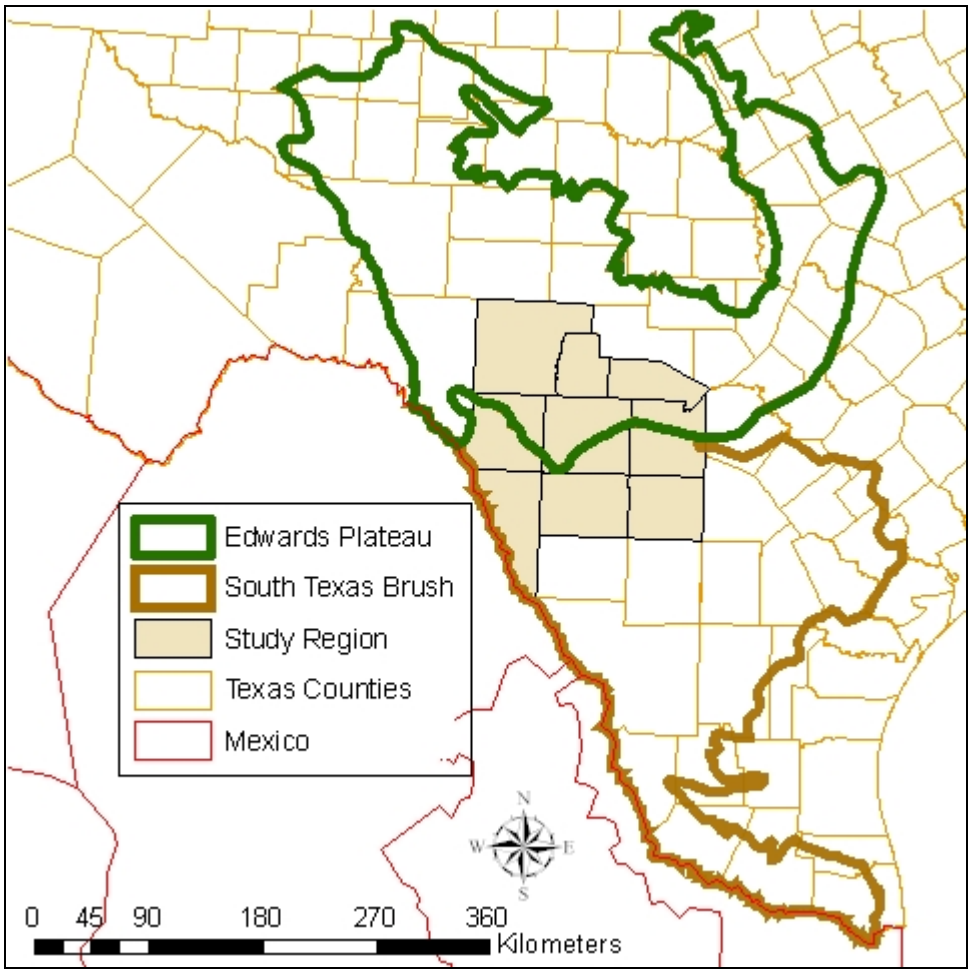

Figure 2. South Texas study region (detailed).

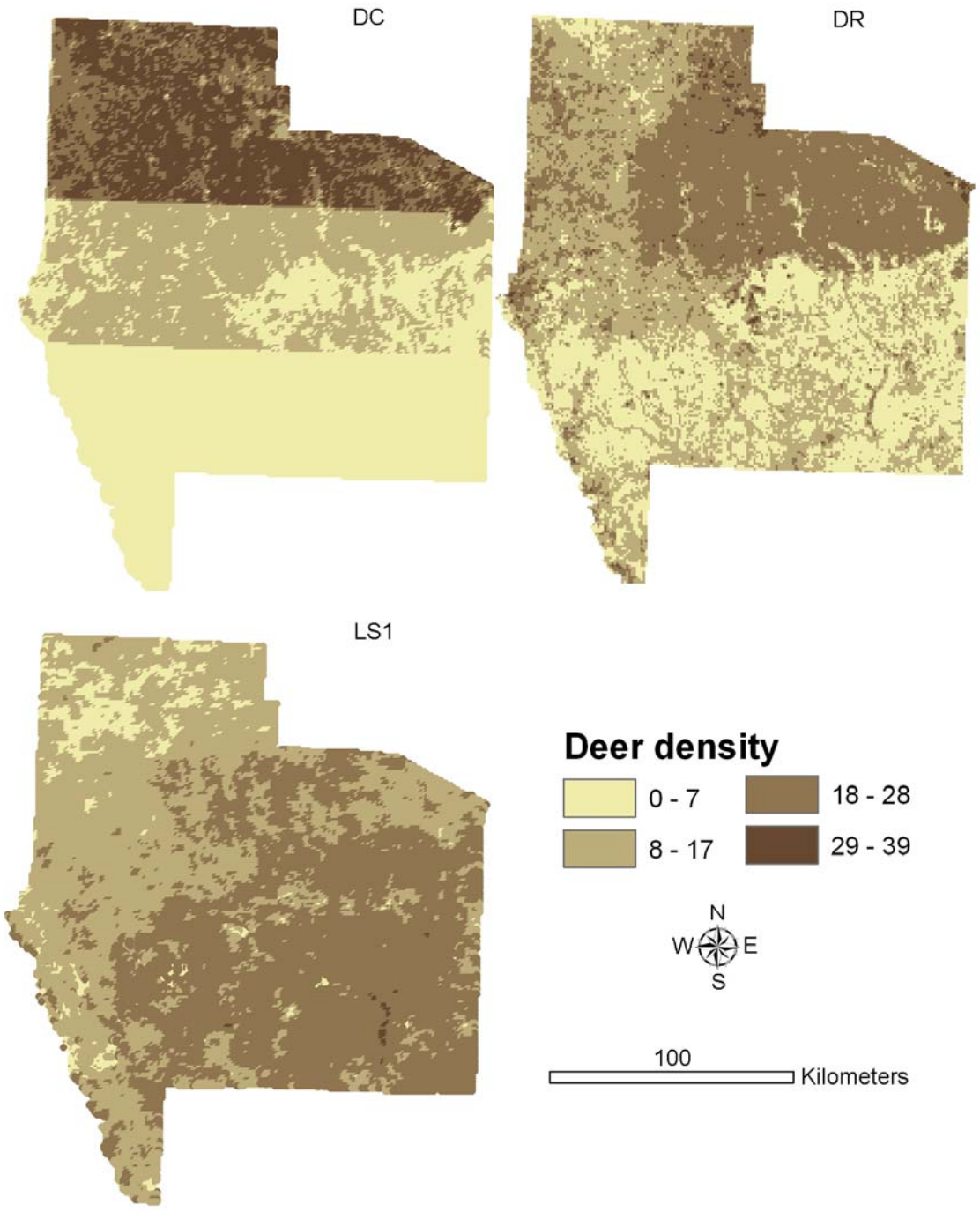

Figure 3. Example surfaces (DC, DR and LS1) from geostatistical estimates of deer distribution and density.

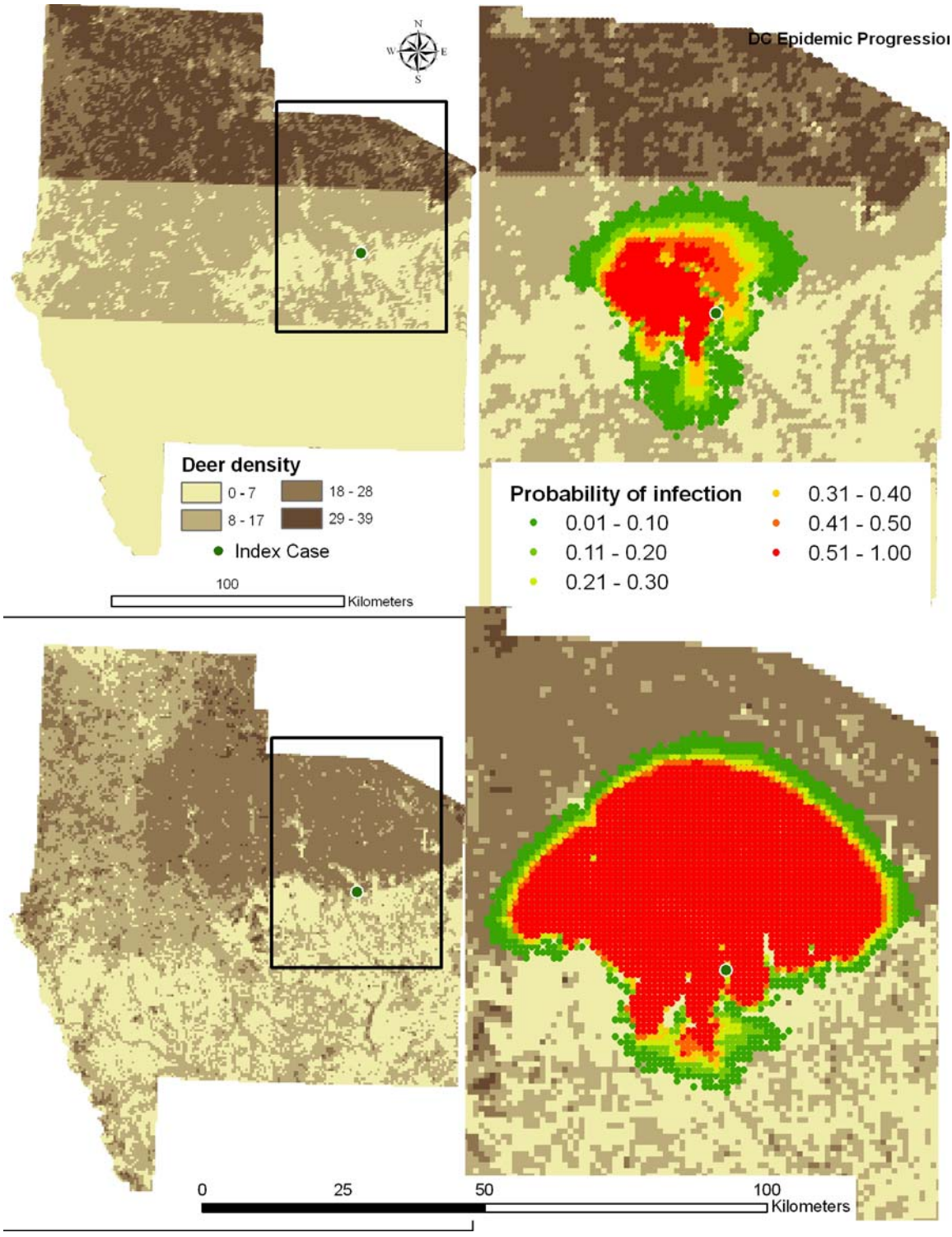

Figure 4. Epidemic progression (DC and DR).

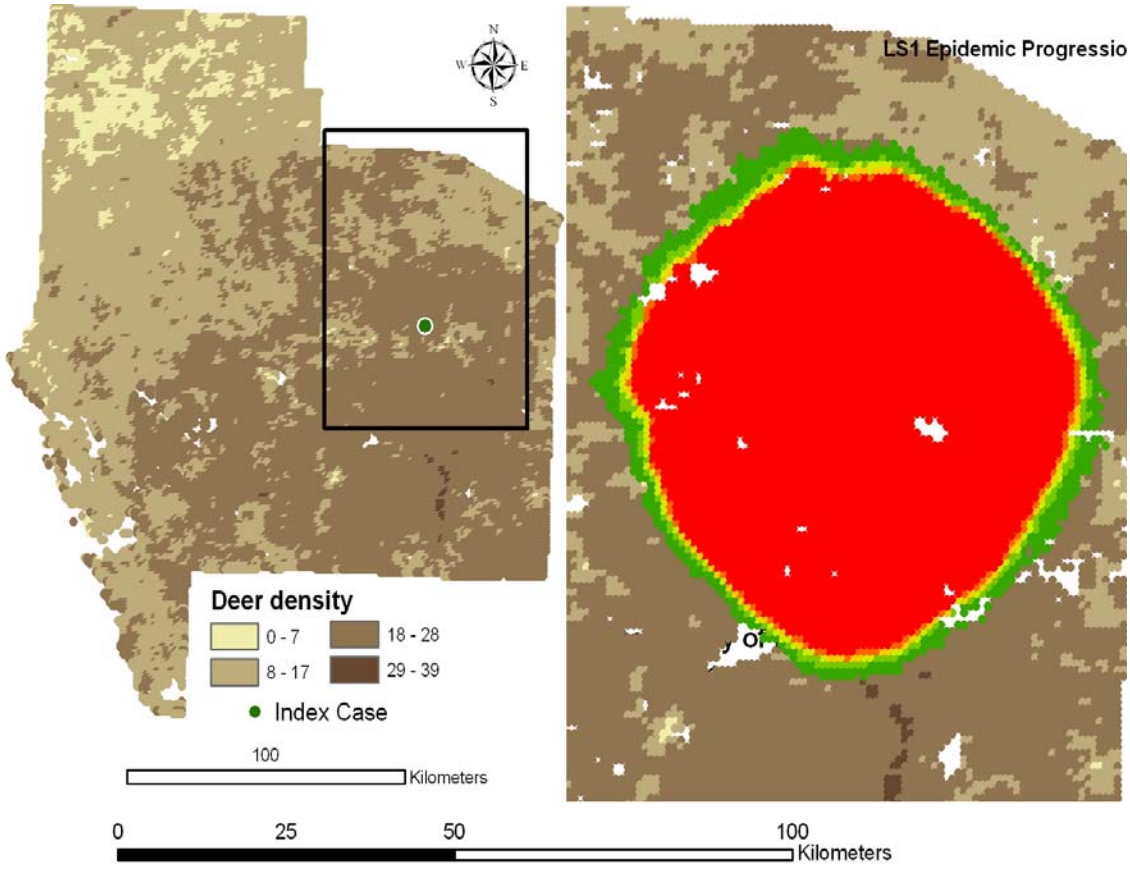

Figure 5. Epidemic progression (LS1).

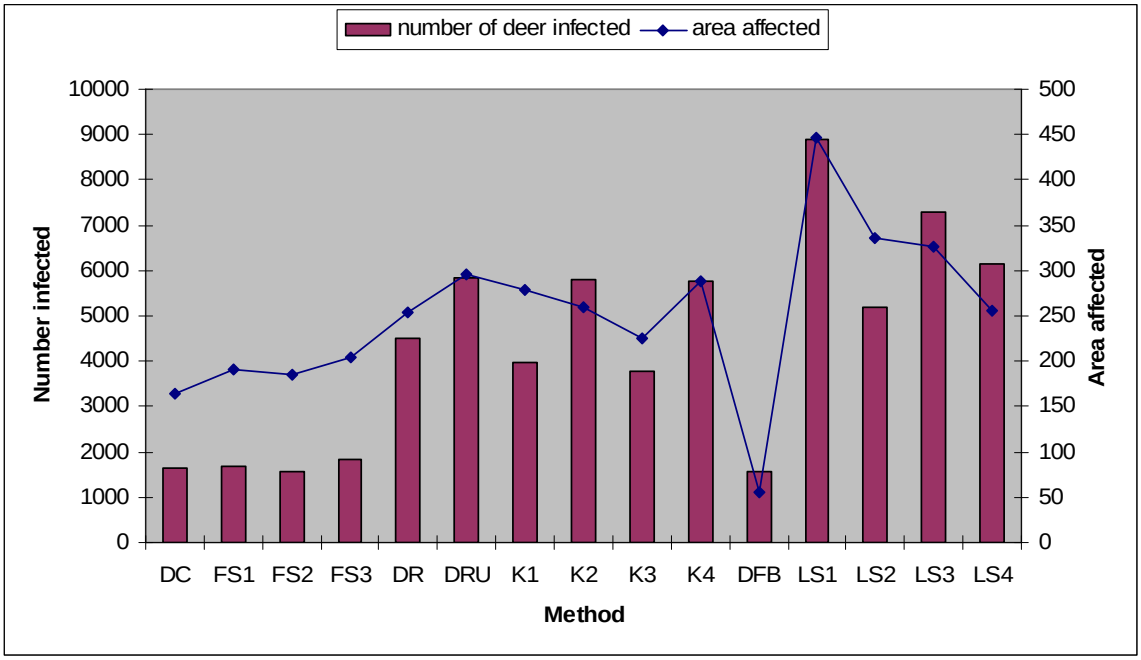

Figure 6. Median number of deer infected and median area affected in km^2 by estimation methodology.

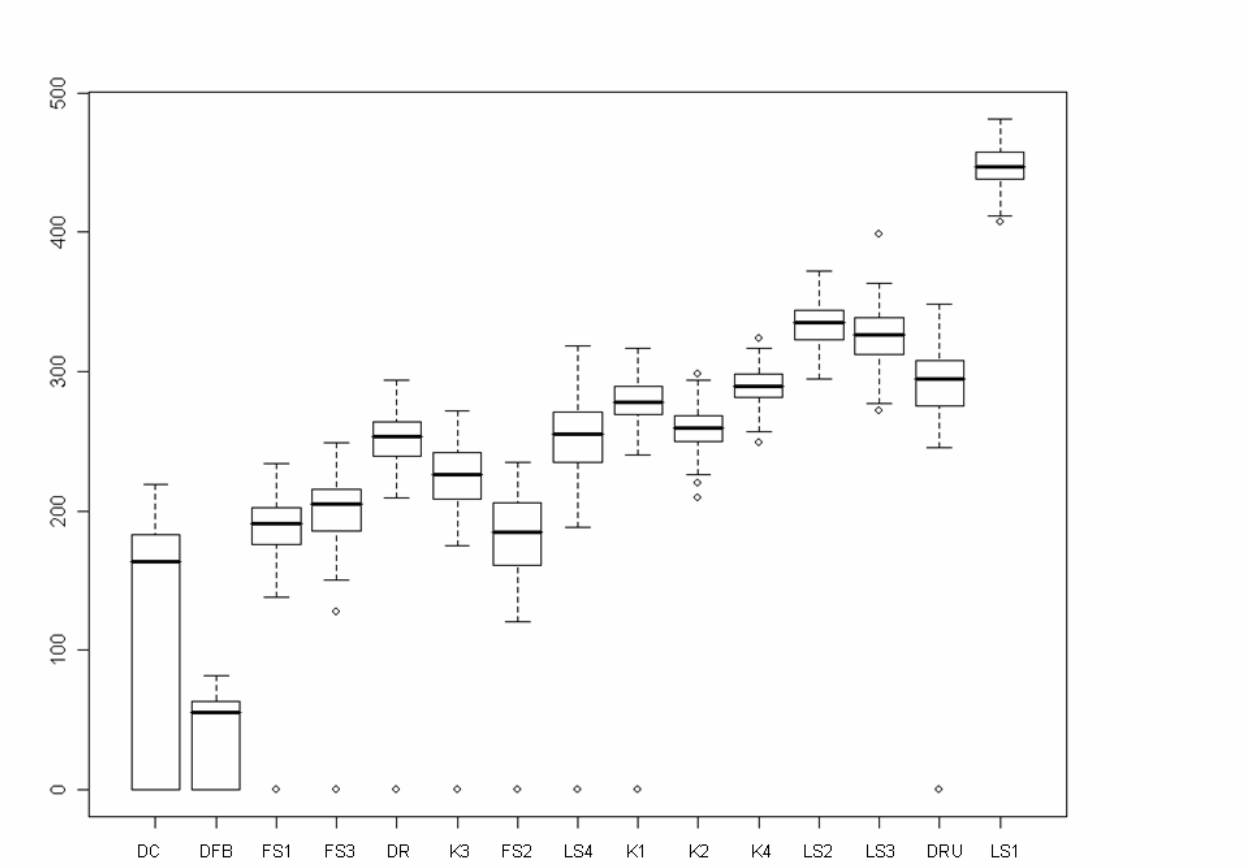

Figure 7. Boxplots of the predicted outbreak distribution for each estimation methodology.

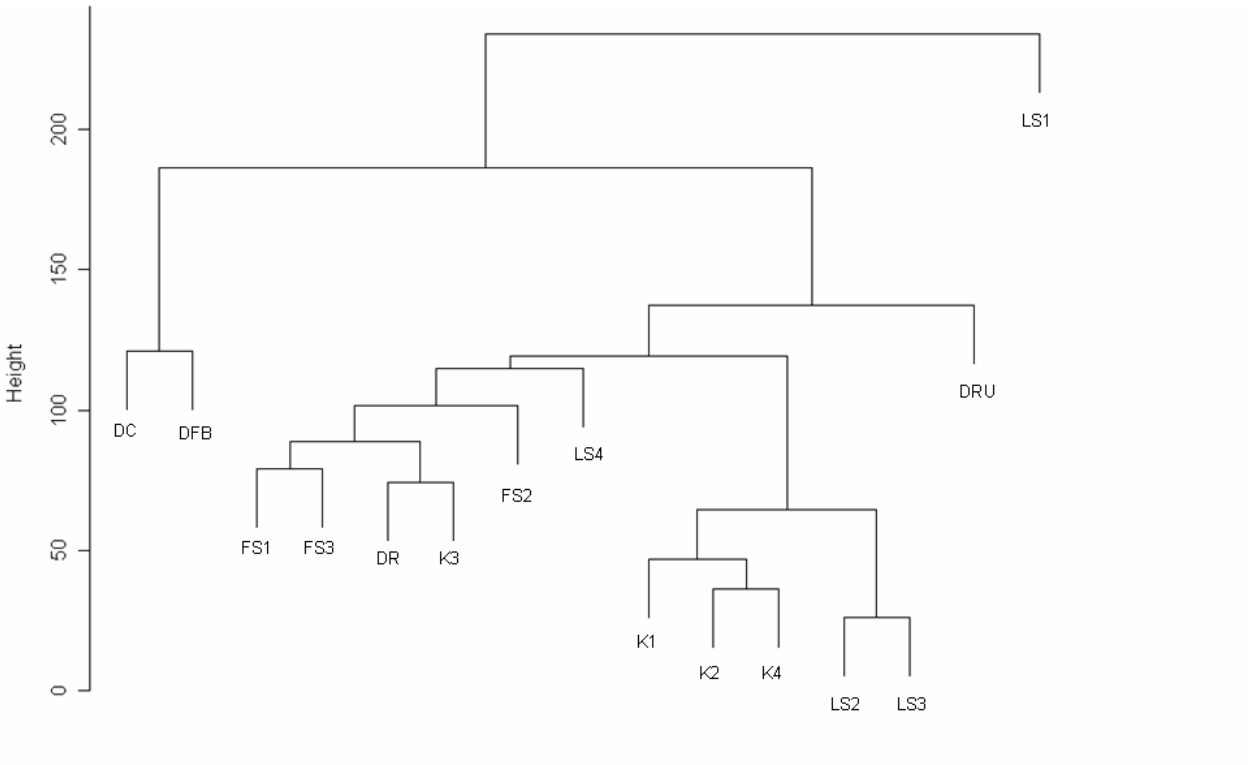

Figure 8. Resulting clusters predicted by hierarchical agglomerative clustering algorithm for the distribution of deer infected.

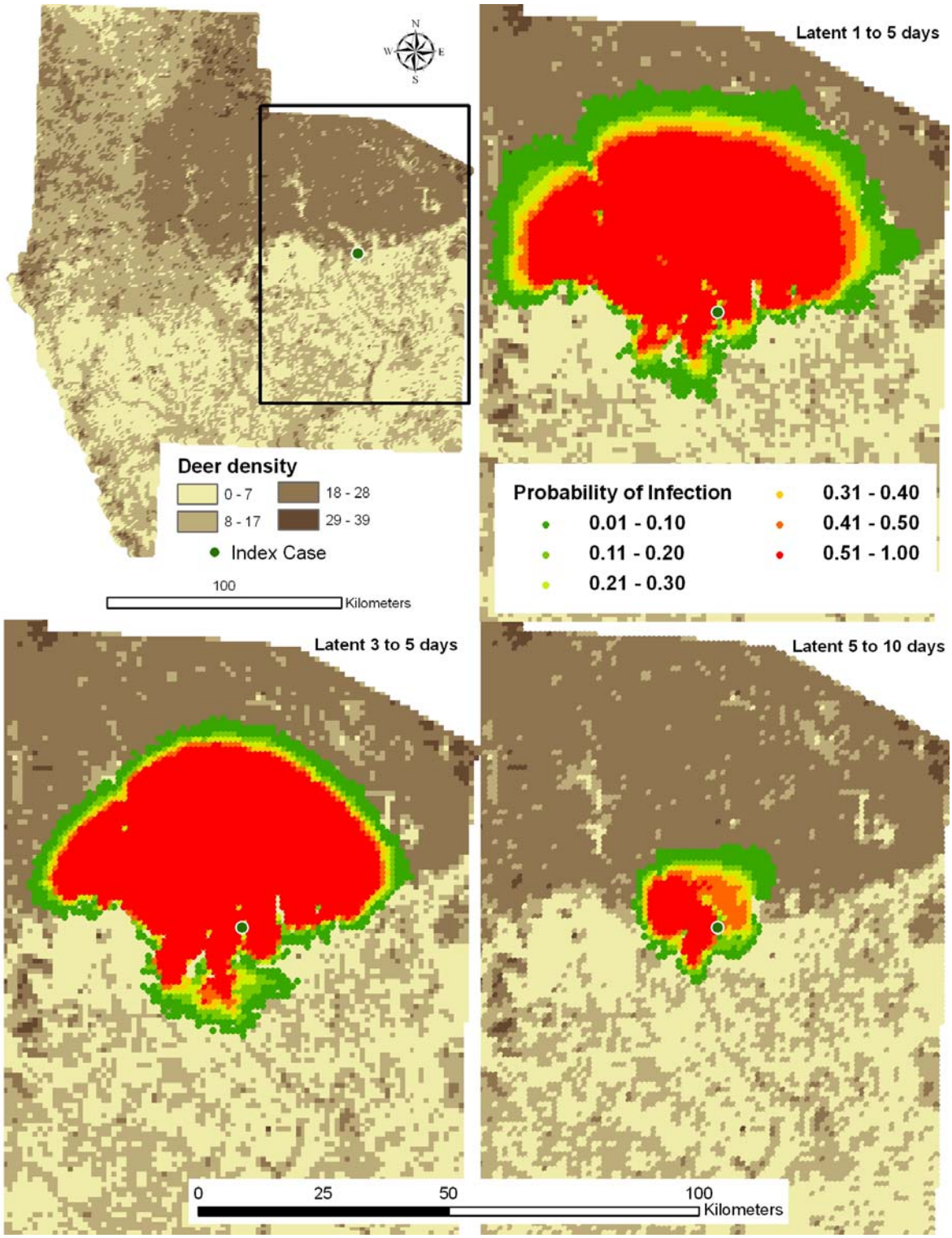

Figure 9. Risk of infection for each spatial location affected for each parameter range for the latent period.

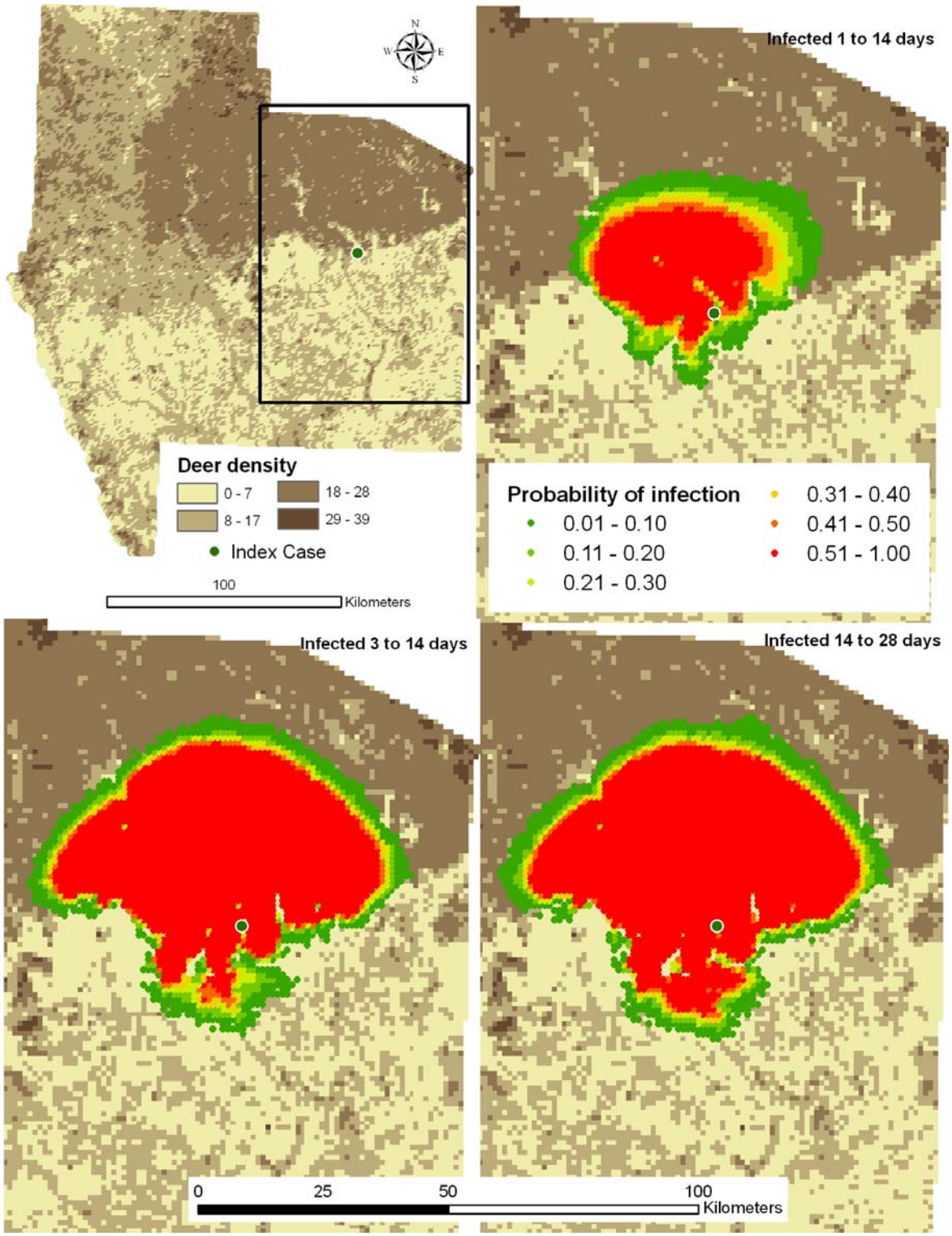

Figure 10. Risk of infection for each spatial location affected for each parameter range for the infectious period.

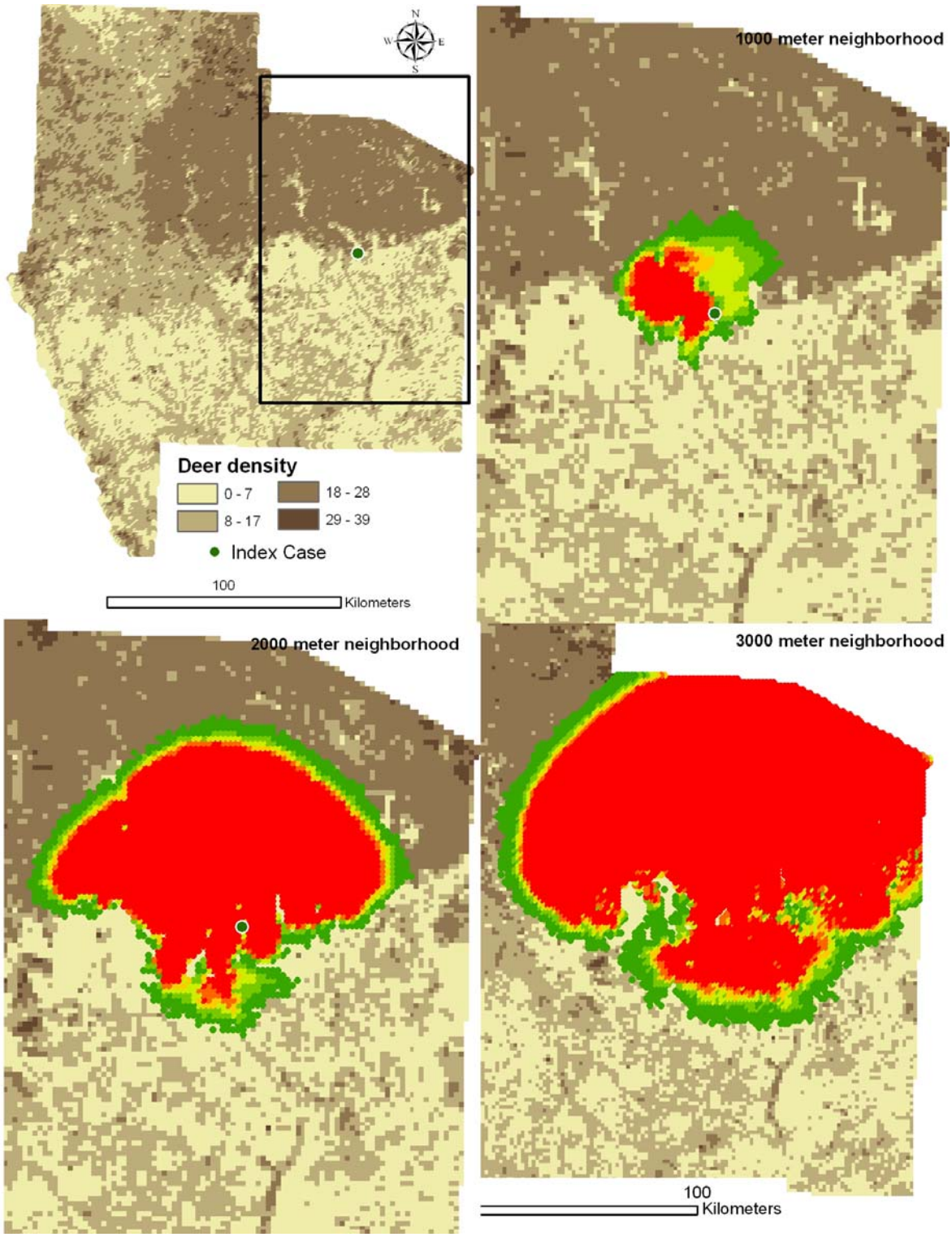

Figure 11. Risk of infection for each spatial location affected for each parameter range for the number of neighbors.

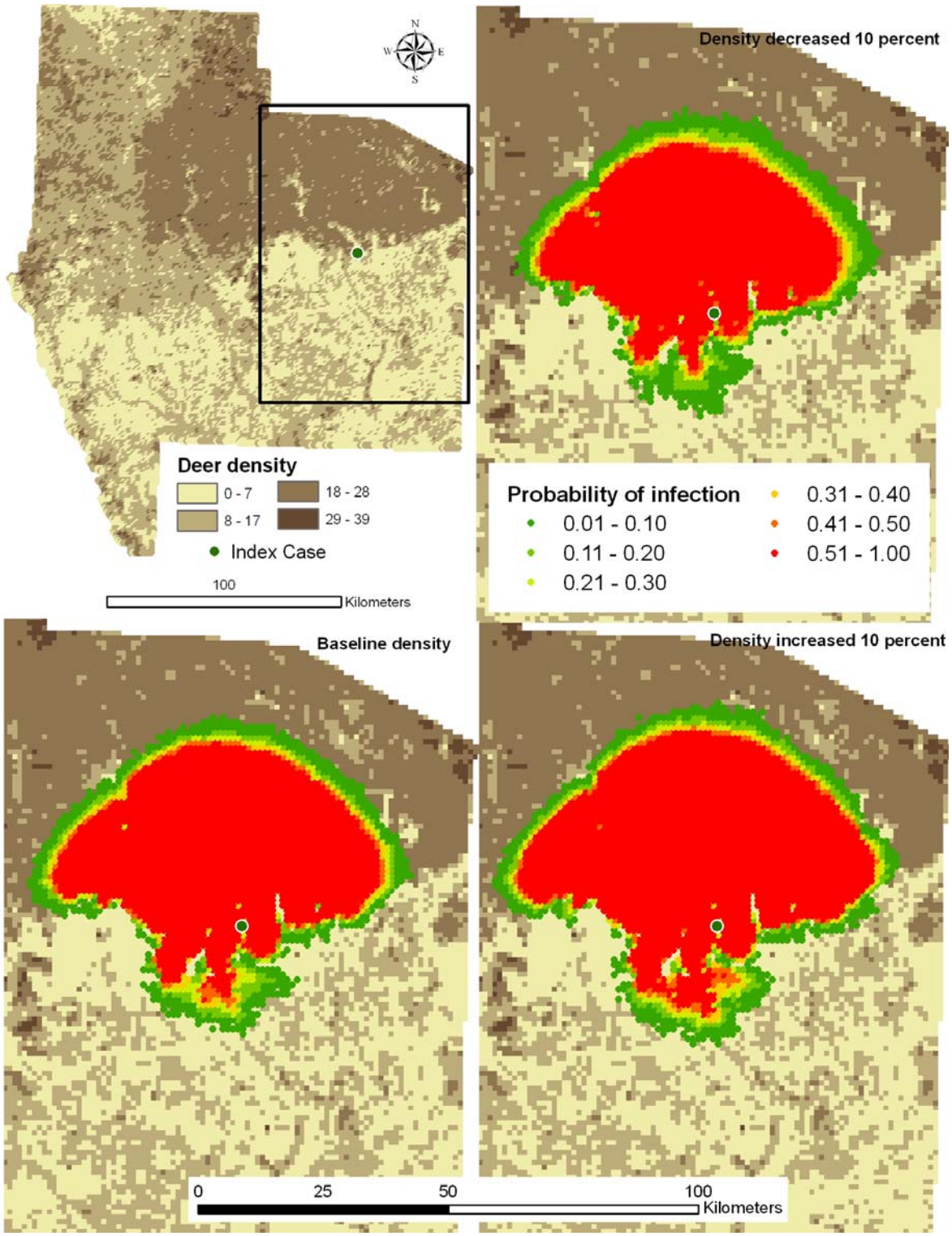

Figure 12. Risk of infection for each spatial location affected for each parameter range for global population density.

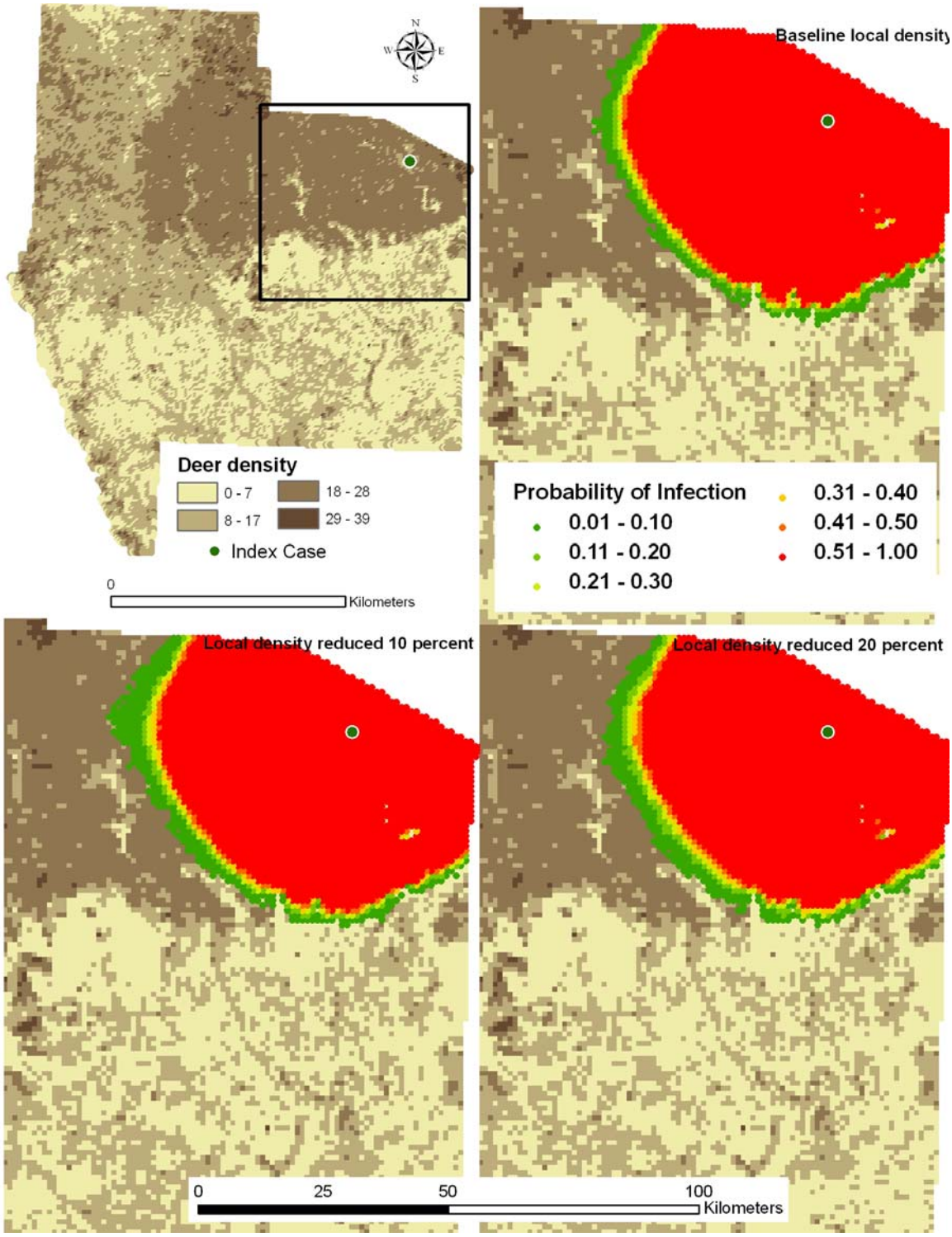

Figure 13. Risk of infection for each spatial location affected for local population density reduction (baseline, 10 percent and 20 percent) within 10 kilometers of a high density index case.

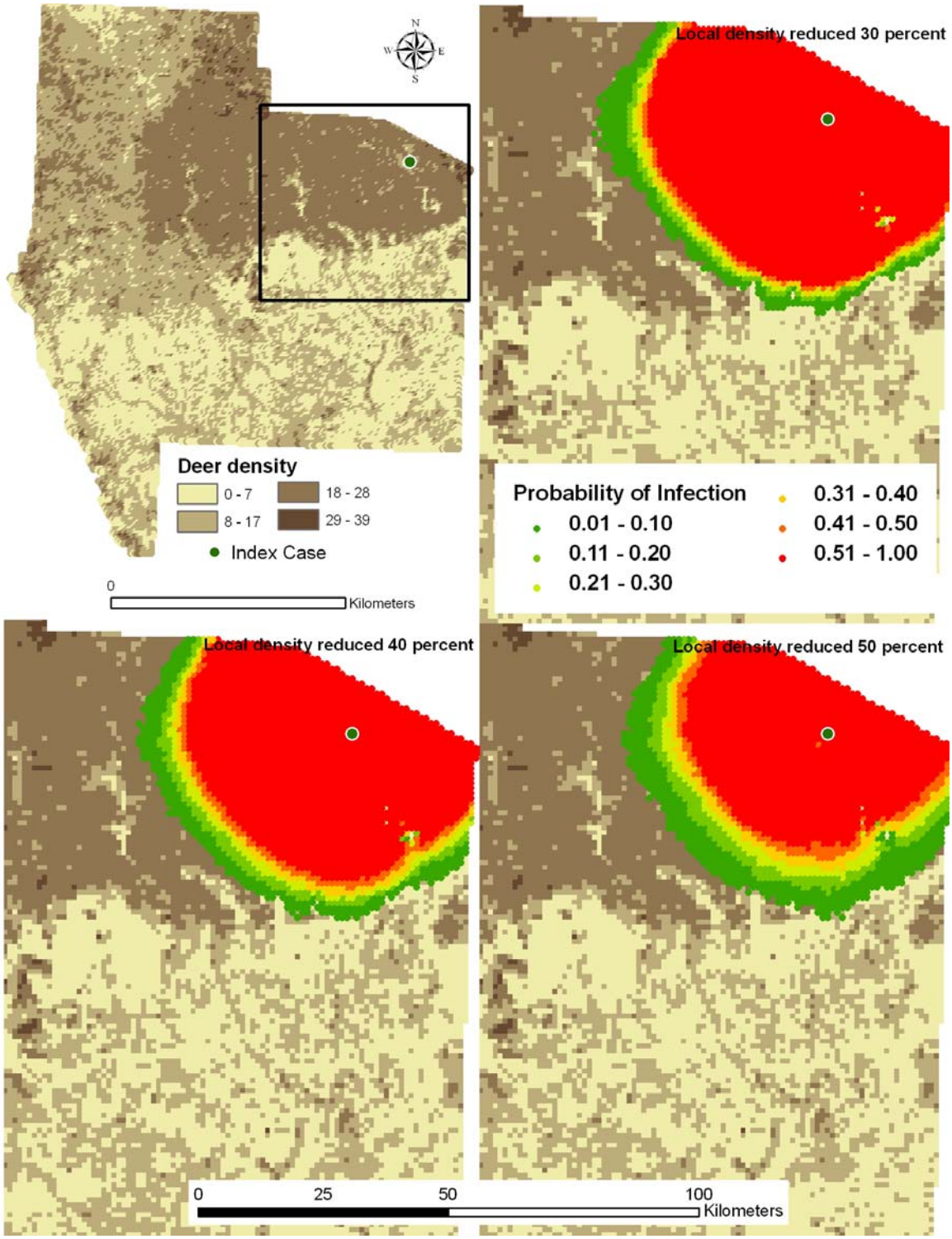

Figure 14. Risk of infection for each spatial location affected for local population density reduction (30 percent, 40 percent and 50 percent) within 10 kilometers of a high density index case.

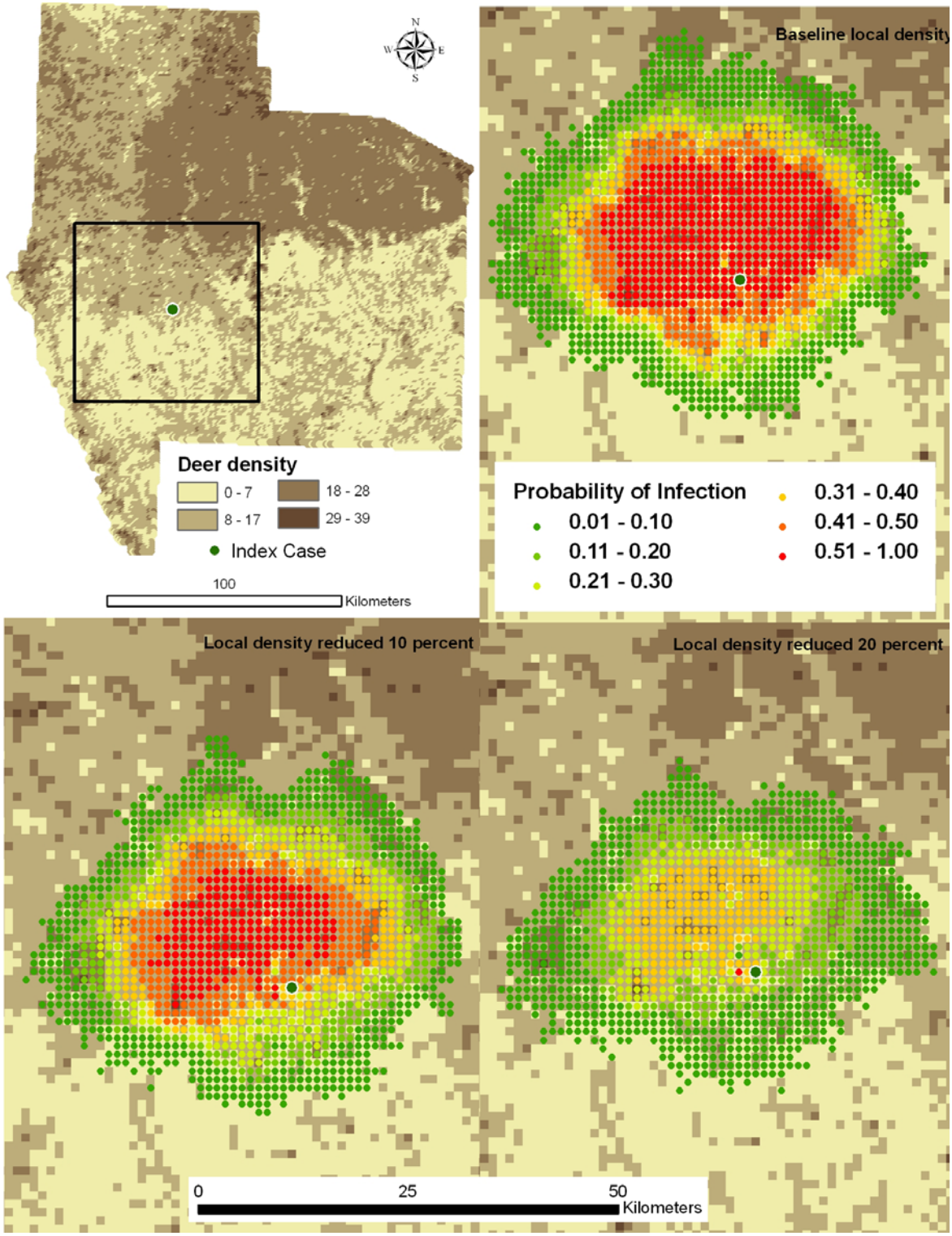

Figure 15. Risk of infection for each spatial location affected for local population density reduction (baseline, 10 percent and 20 percent) within 10 kilometers of a low density index case.

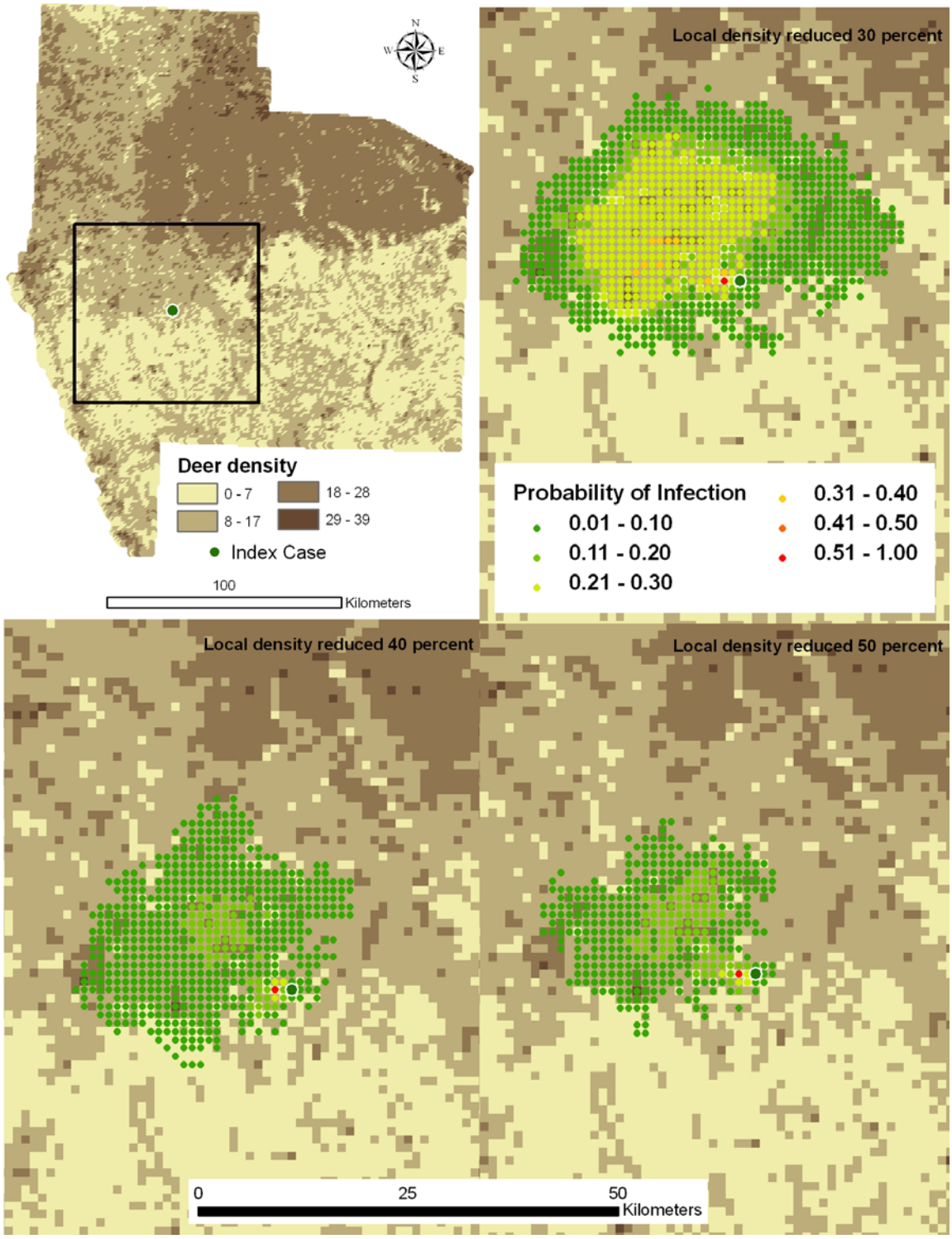

Figure 16. Risk of infection for each spatial location affected for local population density reduction (30 percent, 40 percent and 50 percent) within 10 kilometers of a low density index case.

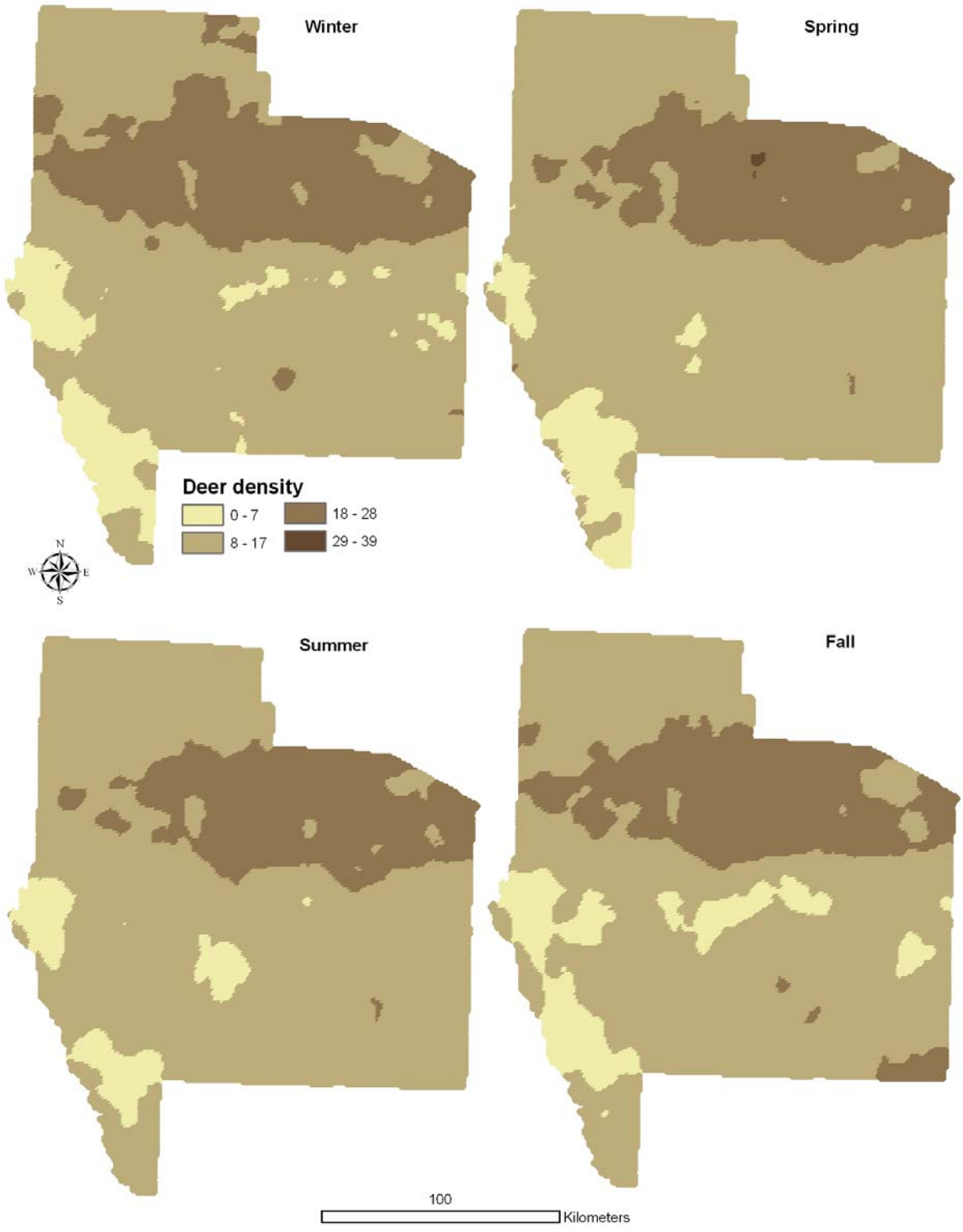

Figure 17. Predicted deer distribution from spatial autoregressive lag models for each season in south Texas.

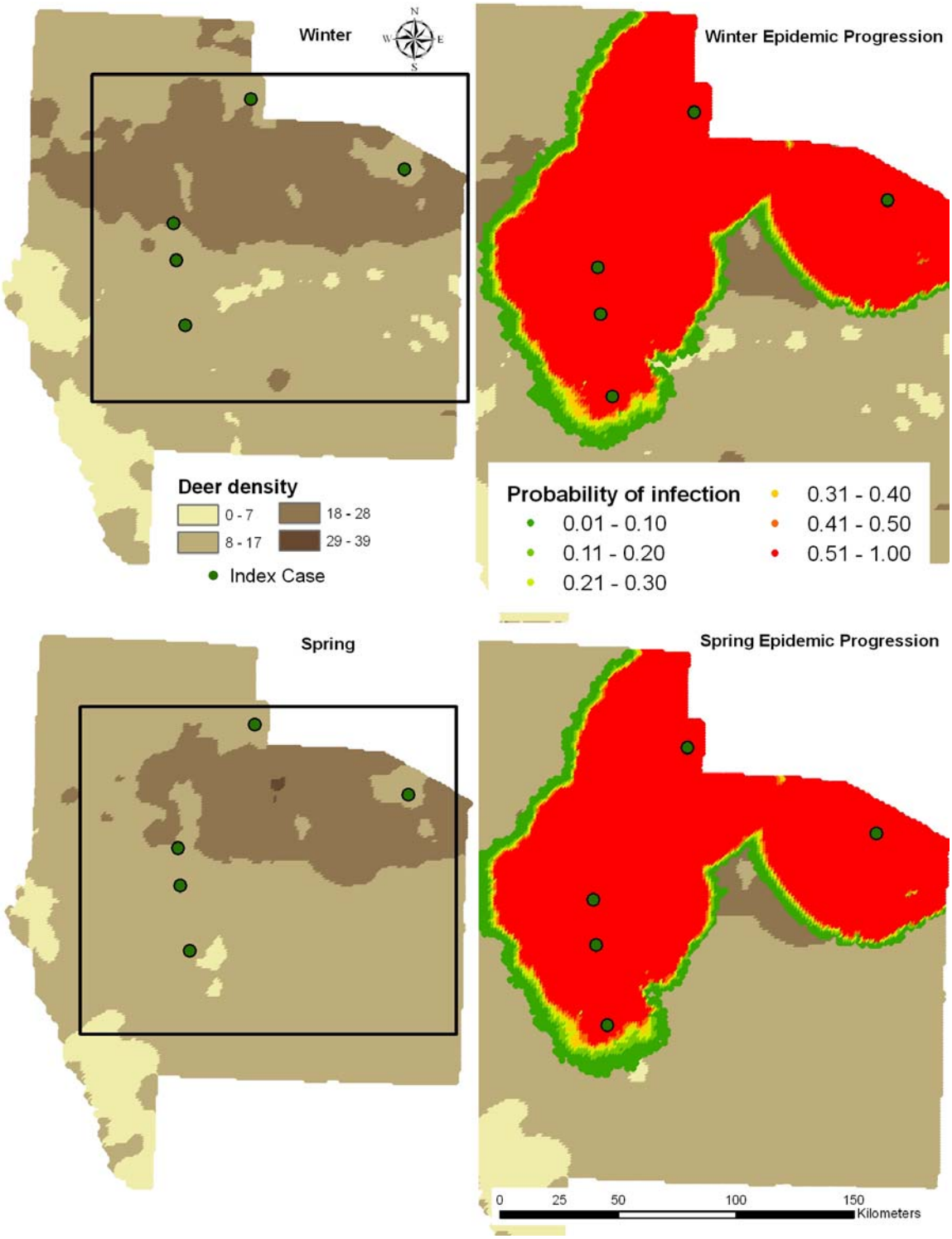

Figure 18. Predicted FMD epidemic progression in the Edwards Plateau Ecoregion of south Texas, for Winter and Spring seasons.

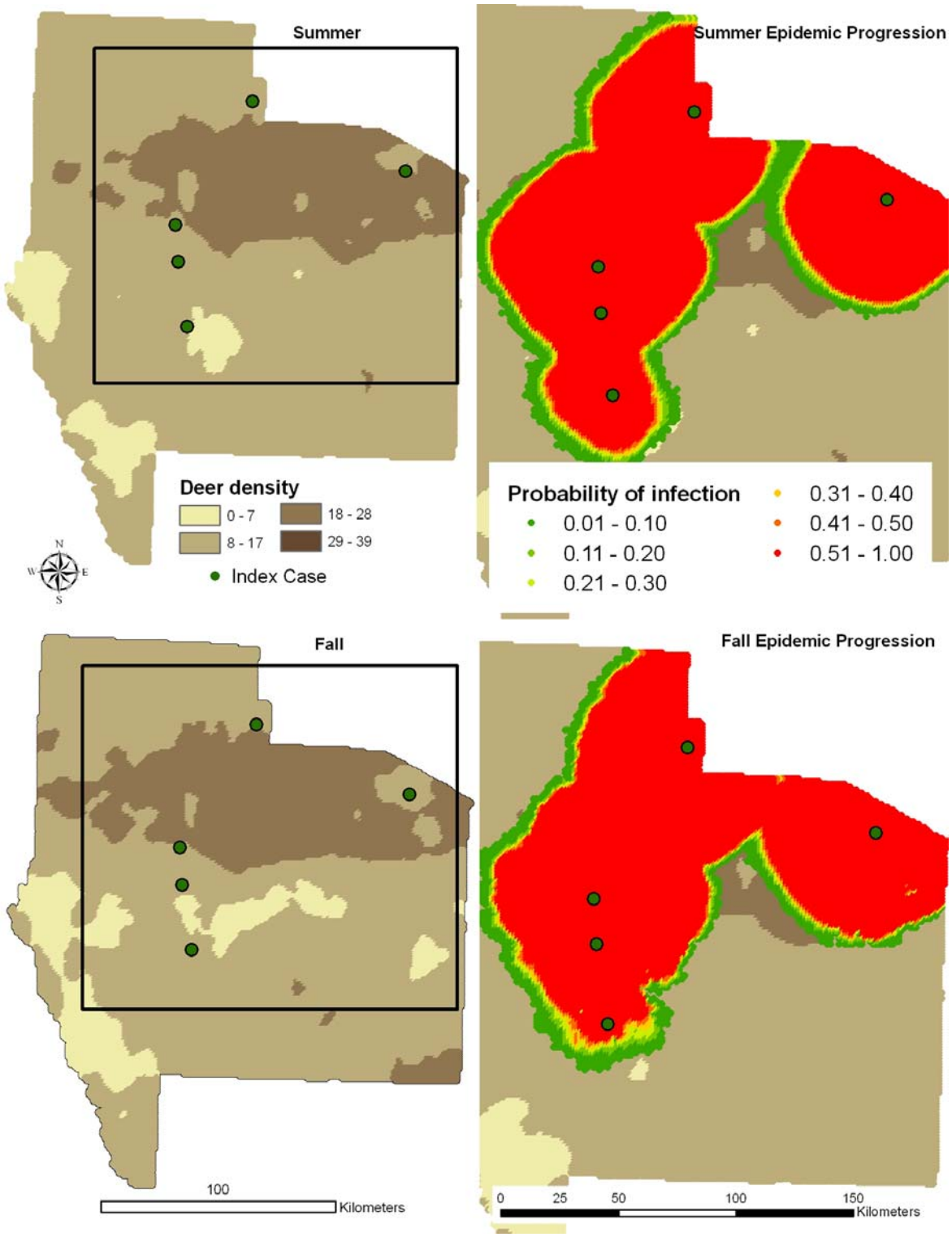

Figure 19. Predicted FMD epidemic progression in the Edwards Plateau Ecoregion of south Texas, for Summer and Fall seasons.

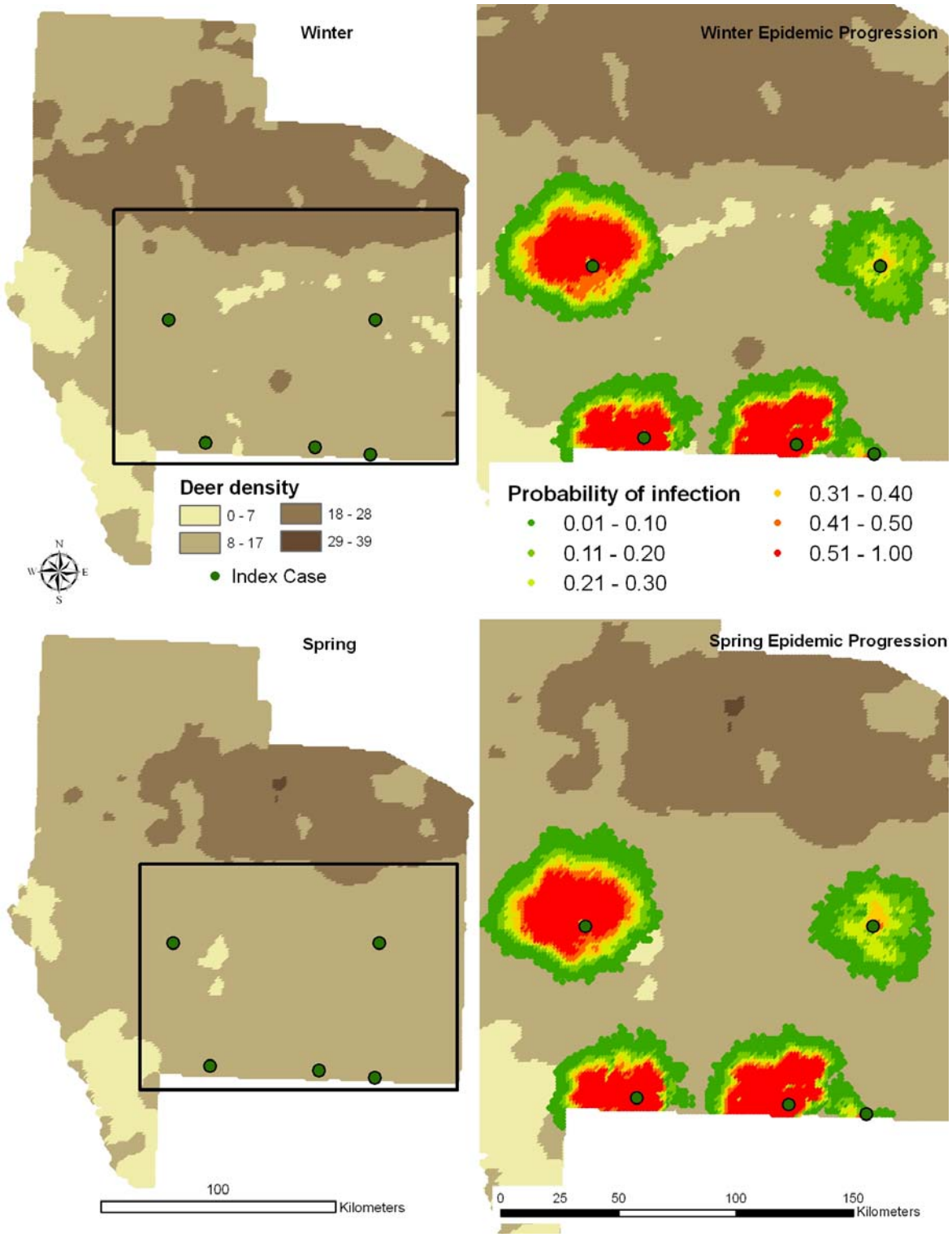

Figure 20. Predicted FMD epidemic progression in the South Texas Brush Ecoregion of south Texas, for Winter and Spring seasons.

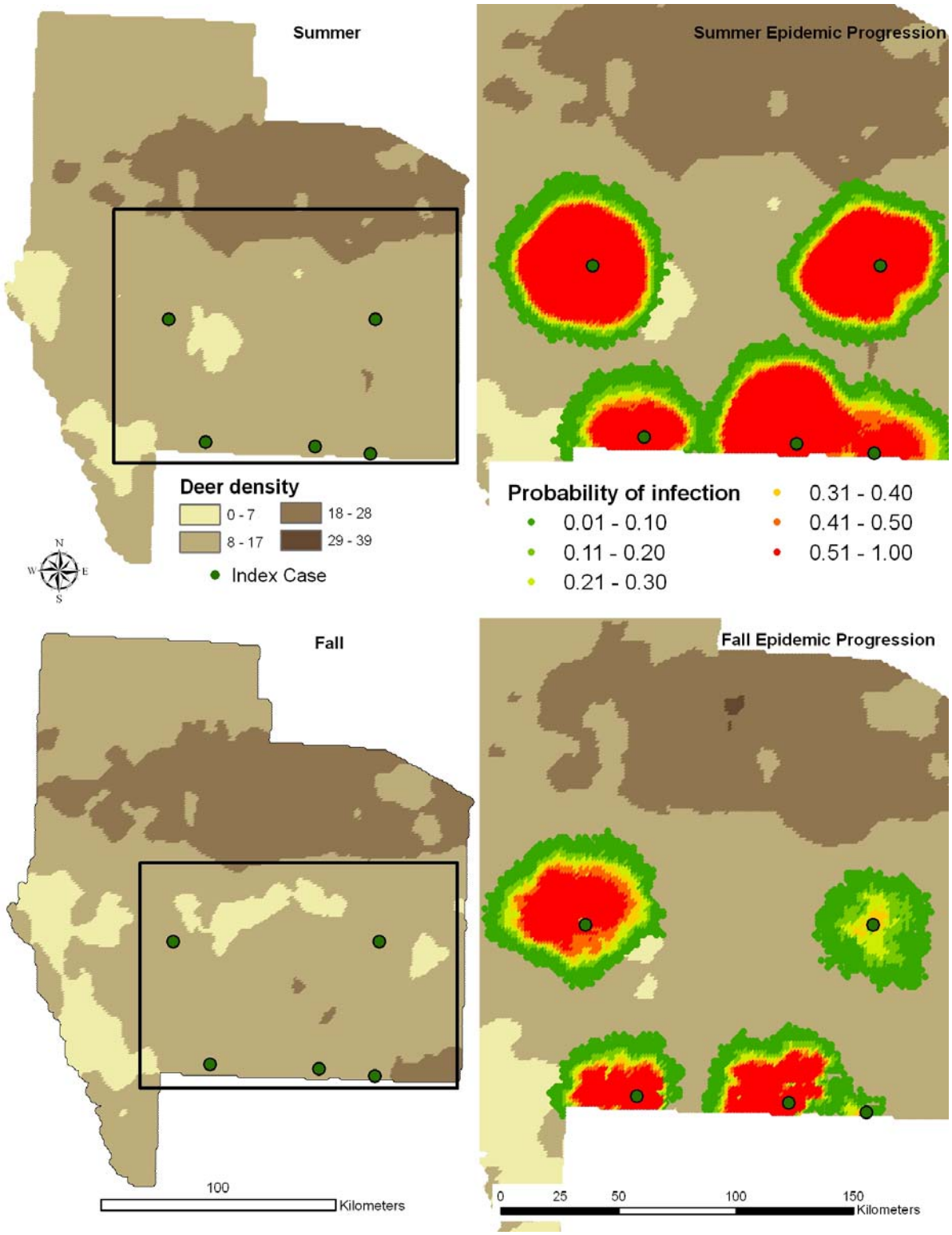

Figure 21. Predicted FMD epidemic progression in the South Texas Brush Ecoregion of south Texas, for Summer and Fall seasons.

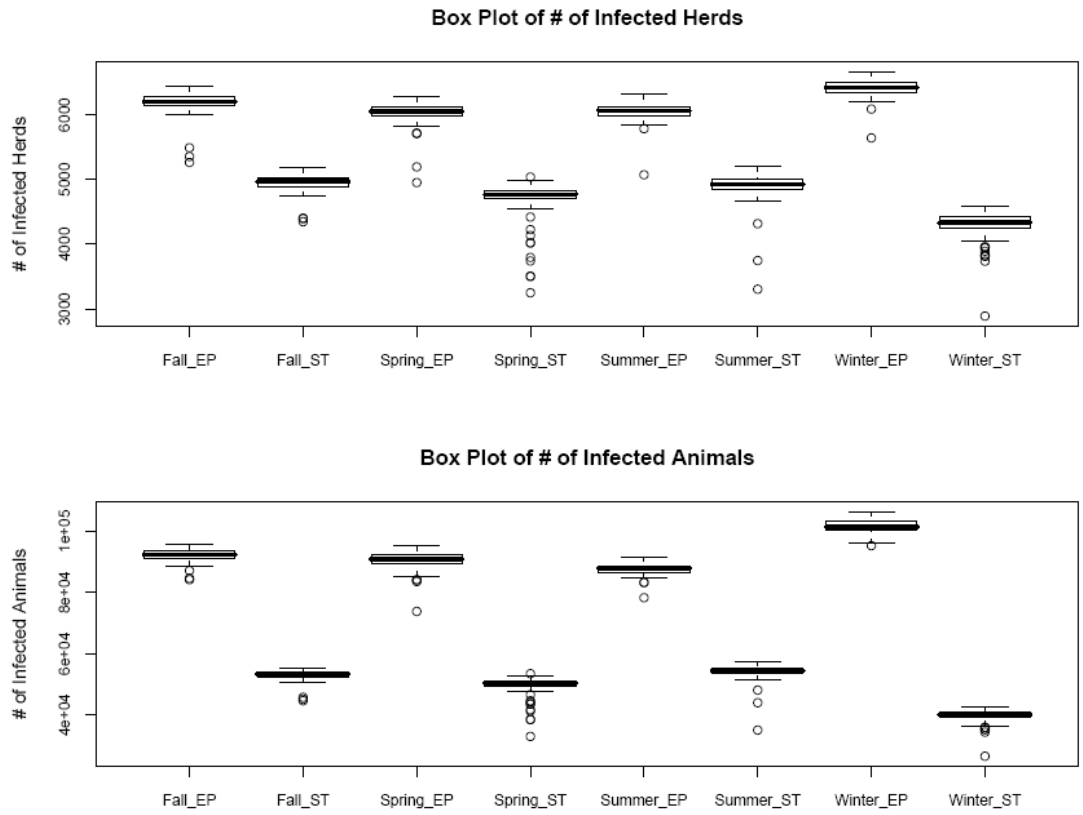

Figure 22. Boxplots of predicted FMD epidemics by season and Ecoregion, infected locations (herds) and infected deer (animals), south Texas.

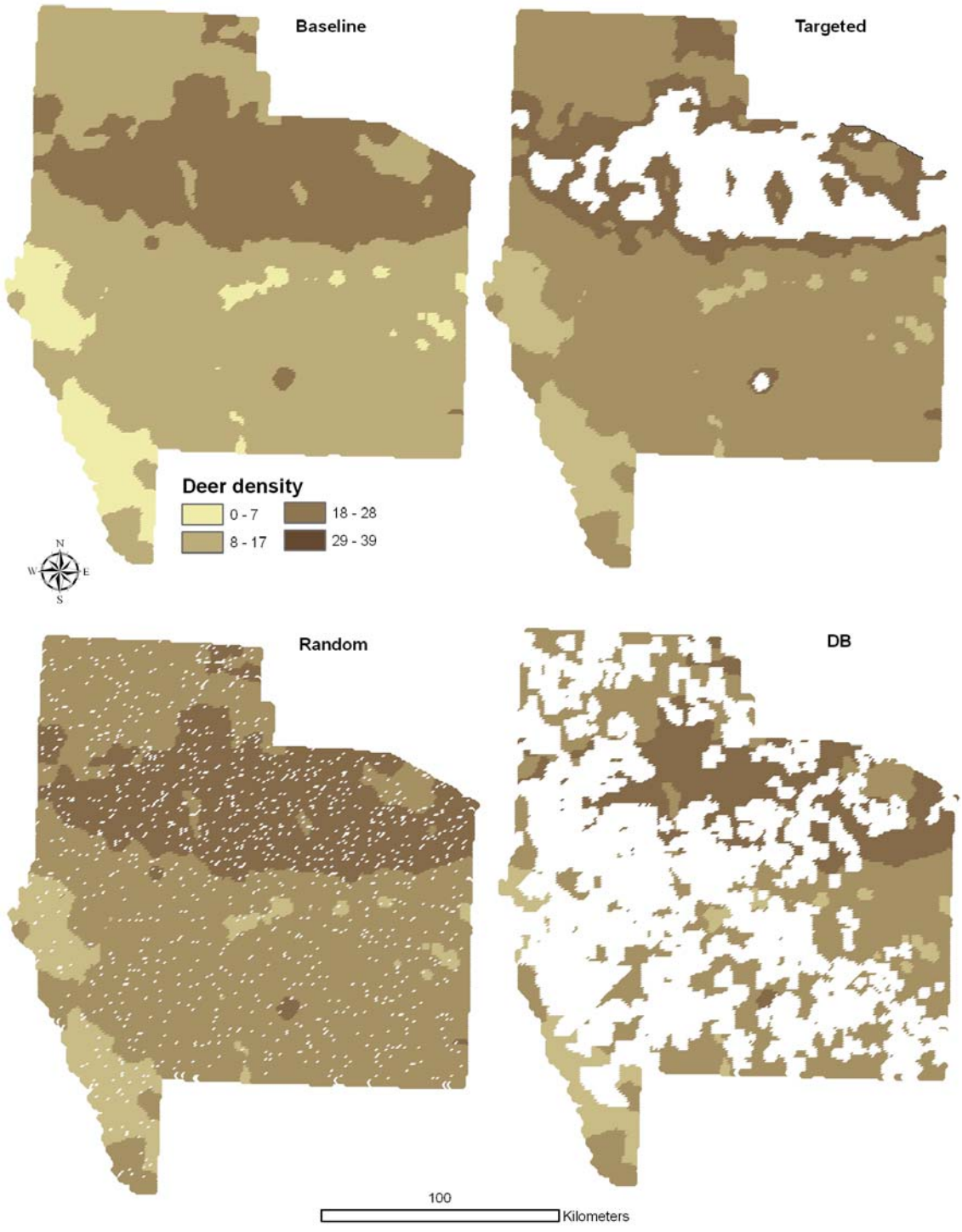

Figure 23. Predicted white tailed deer distributions after application of pre-emptive mitigation strategies (targeted cull, randon cull, depopulation buffer) in an 8-county region of south Texas.

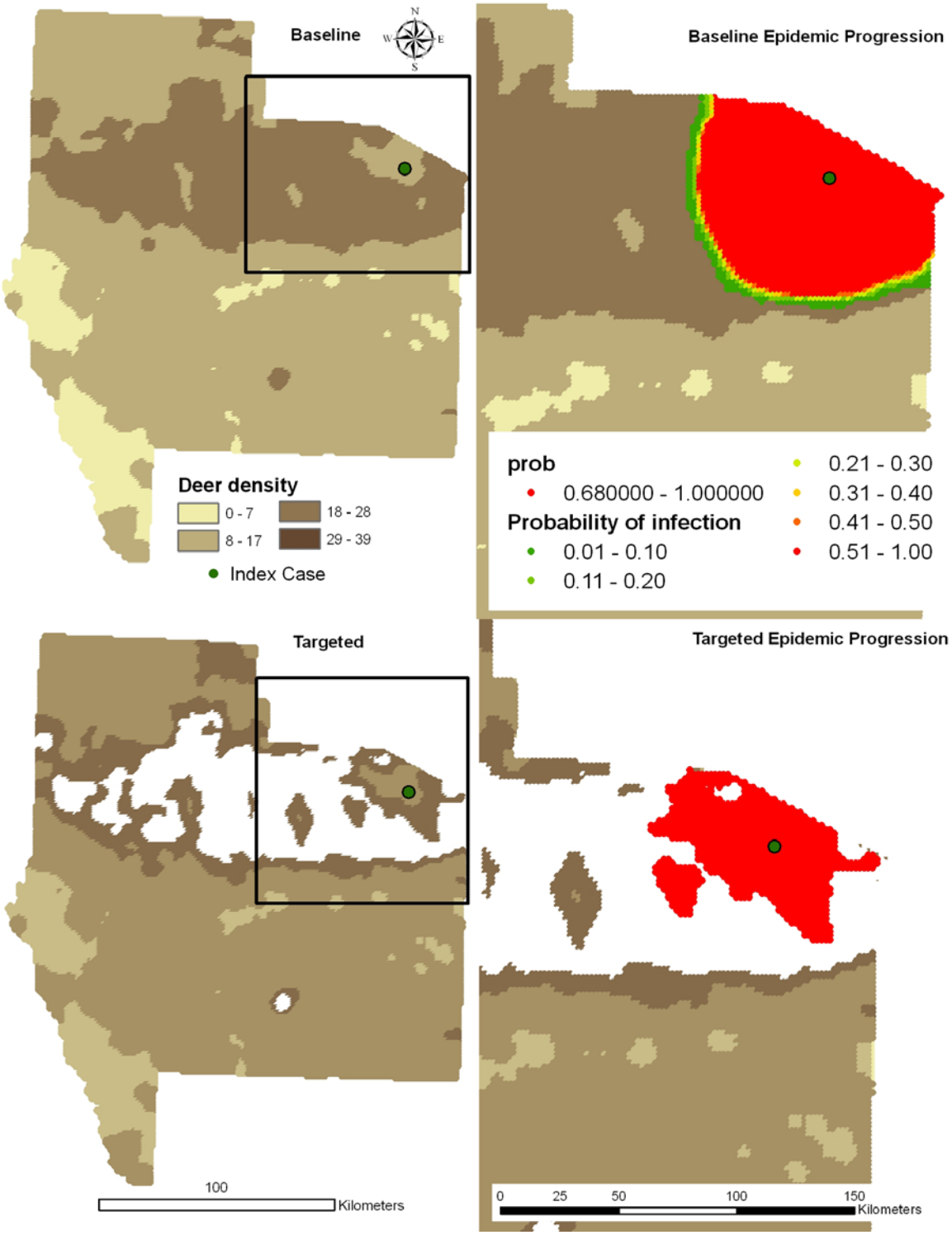

Figure 24. Predicted FMD epidemic progression in white tailed deer populations within the Edwards Plateau Ecoregion of an 8-county region of south Texas, by mitigation strategies (baseline, targeted cull).

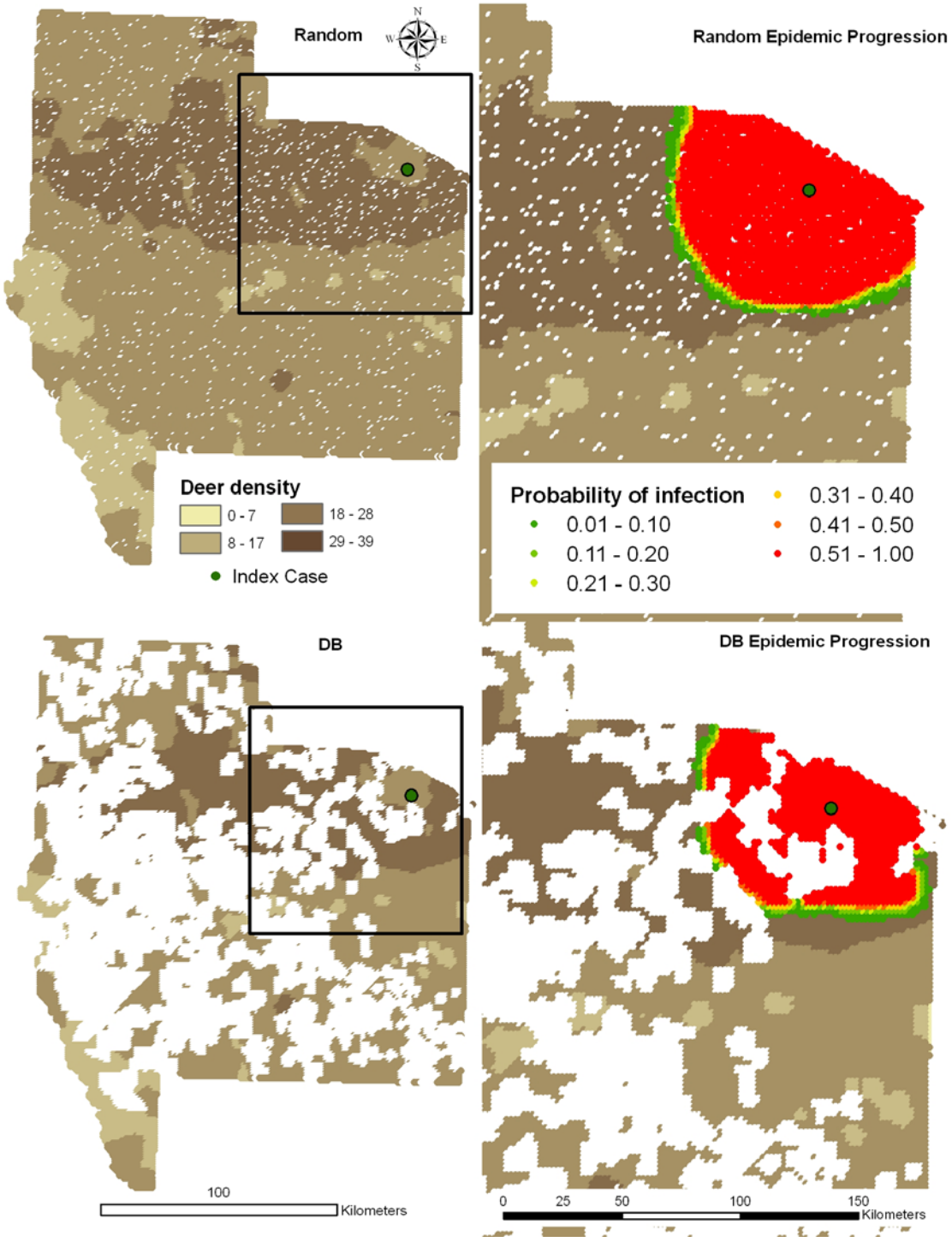

Figure 25. Predicted FMD epidemic progression in white tailed deer populations within the Edwards Plateau Ecoregion of an 8-county region of south Texas, by mitigation strategies (random, depopulation buffer).

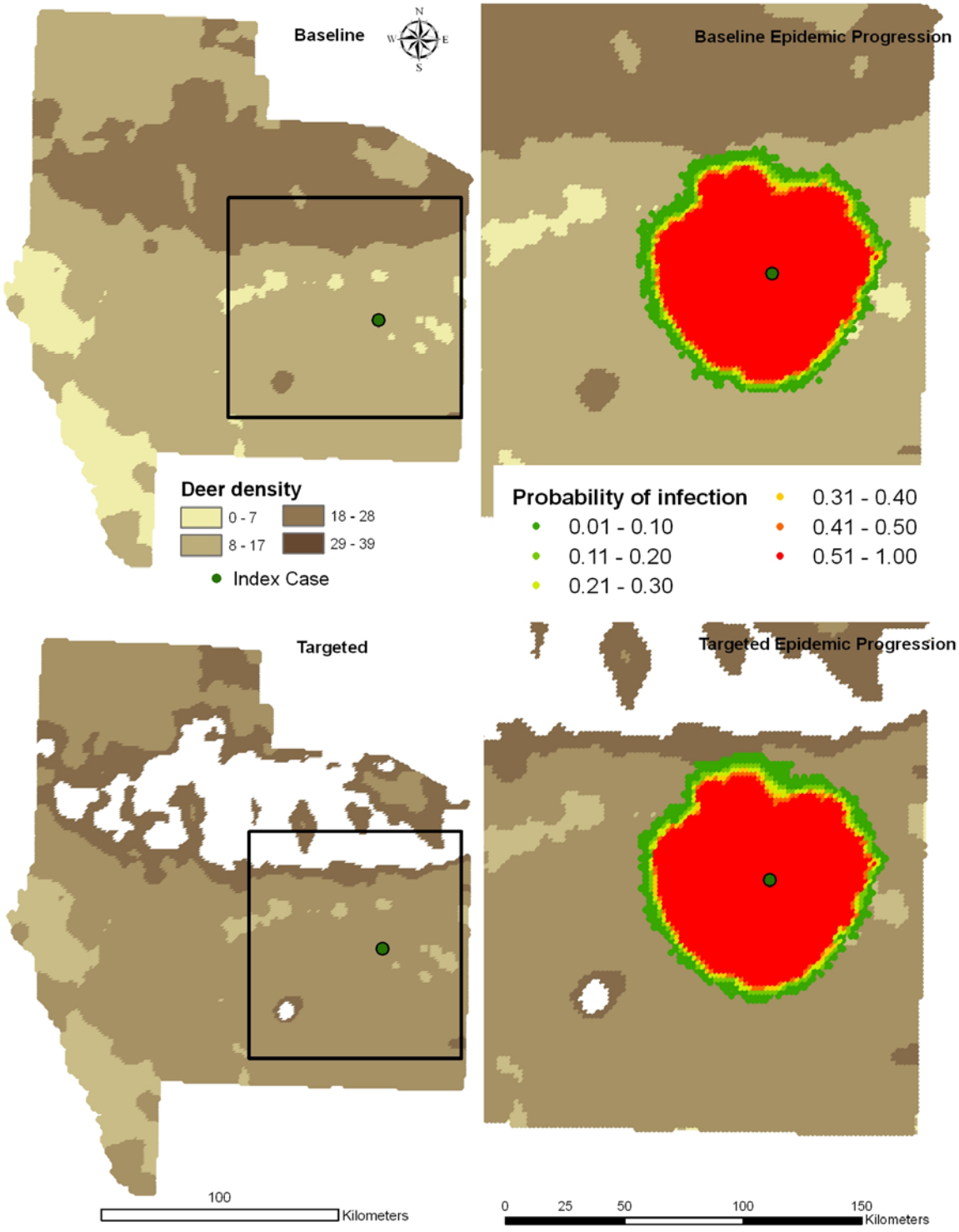

Figure 26. Predicted FMD epidemic progression in white tailed deer populations within the South Texas Brush Ecoregion of an 8-county region of south Texas, by mitigation strategies (baseline, targeted cull).

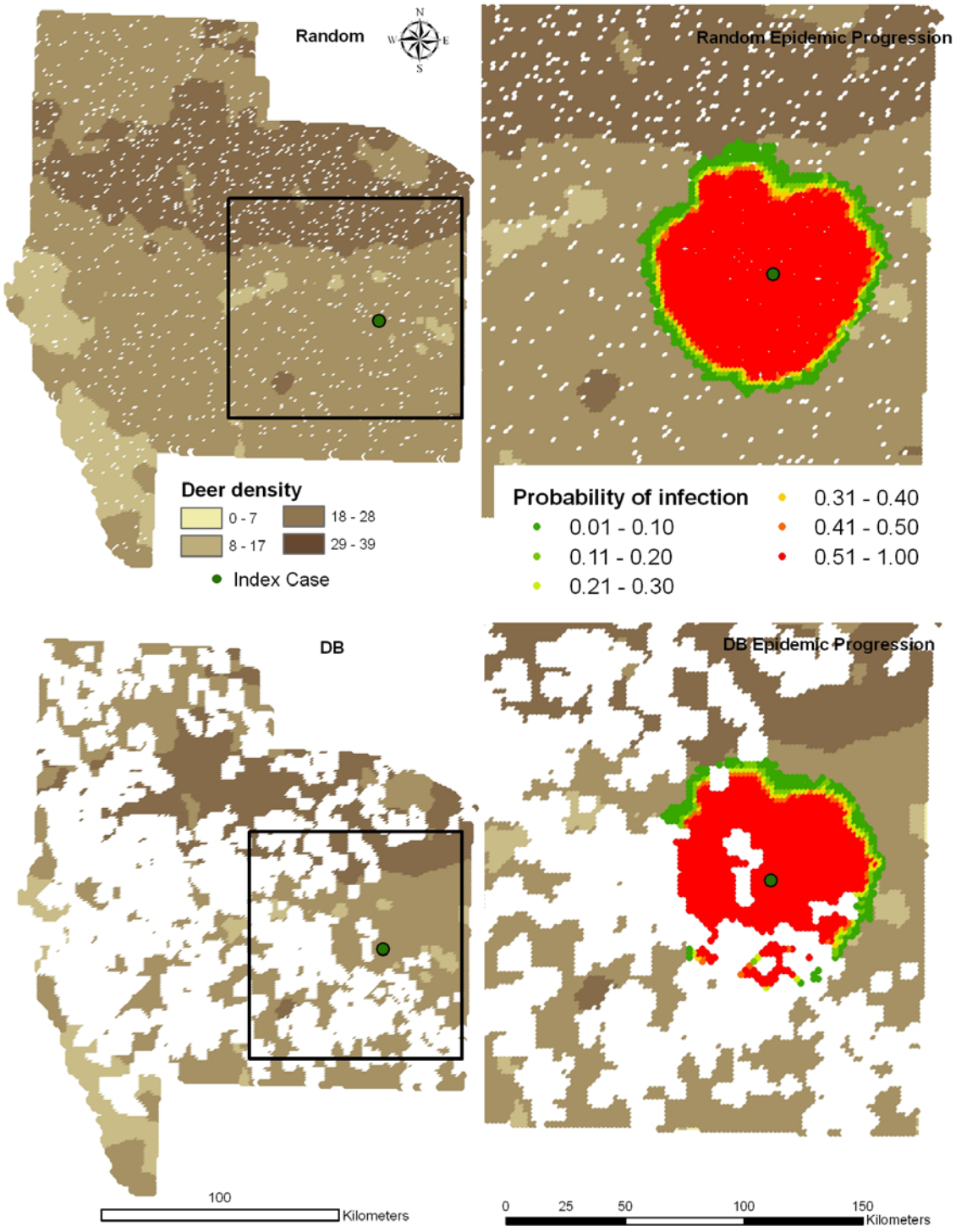

Figure 27. Predicted FMD epidemic progression in white tailed deer populations within the South Texas Brush Ecoregion of an 8-county region of south Texas, by mitigation strategies (random, depopulation buffer).

APPENDIX C

TABLES

Table 1. Count and percentage of suitable pixels per land use category.

Land Use Category	Count	Percentage
Forest	7692254	0.27
Shrub	16774976	0.14
Grassland	4049744	0.59
Total	28516974	100

Table 2. Predicted number of deer by estimation methodology as compared to Texas Parks and Wildlife Department provided county estimate.

	Number of deer	
Method	Estimated	TPWD (n=427,292) % estimated
DC	412,770	97
FS1	395,281	93
FS2	395,828	93
FS3	396,269	93
DR	410,624	96
DRU	768, 493	179
K1	403, 027	94
K2	439,169	103
K3	536,042	125
K4	385,939	90
DFB	385,939	90
LS1	491,566	115
LS2	415,426	97
LS3	470,428	110
LS4	464,680	109

Table 3. Predicted size of an outbreak of foot and mouth disease in a population of deer in southern Texas for each estimation method. Results shown are from 100 simulations of the geographic automata model for each deer surface.

Method	Number of deer					Area (km^2)				
	Median	IQR	5%, 95%	Skewness	Kurtosis	Median	IQR	5%, 95%	Skewness	Kurtosis
DC	1626	1830	0, 2051	-0.847	-1.095	164	182	0, 205	-0.849	-1.095
FS1	1661	263	0, 1935	-2.59	6.438	191	27	0, 219	-0.2711	-6.907
FS2	1563	439	0, 1931	-1.512	1.011	185	45	0, 226	-1.601	-1.165
FS3	1839	344	0, 2155	-2.198	4.4	205	30	0, 229	-2.505	5.538
DR	4510	429	0, 4991	-3.065	8.415	253	25	0, 281	-3.072	8.426
DRU	5829	724	0, 6570	-1.59	0.719	295	31	0, 326	-1.61	0.745
K1	3978	297	3515, 4373	-5.265	32.385	278	20	248, 302	-5.574	34.95
K2	5798	492	5110, 6331	-0.678	1.625	259	17	234, 286	-0.098	0.792
K3	3791	689	3028, 4416	-2.894	17.993	226	33	188, 255	-3.938	27.66
K4	5752	562	5225, 6351	0.059	-0.773	289	17	266, 311	-0.16	-0.09
DFB	1572	1899	0, 2340	-0.436	-1.329	56	63	0, 76	-0.551	-1.399
LS1	8896	374	8493, 9381	-0.075	0.173	447	19	427, 471	-0.088	0.198
LS2	5190	349	4762, 5443	-0.227	-0.39	335	21	303, 353	-0.317	0.026
LS3	7304	589	6639, 8039	-0.267	1.088	326	27	291, 354	-0.047	1.107
LS4	6149	1231	0, 7080	-1.773	1.701	255	38	0, 294	-1.862	1.922

Table 4. Predicted number of deer infected from an outbreak of foot and mouth disease in a population of deer in southern Texas for each latent period modeled as a uniform probability distribution. Results shown are from 100 simulations of the geographic automata model using the baseline deer surface.

Latent period	Number of deer					
	Median	% change, baseline	IQR	5%, 95%	Skewness	Kurtosis
1 – 5 days	79242	105.63%	11729	5, 91424	-2.99	11.51
3 – 5 days	**38537**	**NA**	**2781**	**34785, 41829**	**-0.431**	**4.53**
5 – 10 days	3779	-90.19%	4658	5, 5480	-0.432	-1.804

Parameters and results in bold are from the baseline scenario

Table 5. Predicted number of locations infected from an outbreak of foot and mouth disease in a population of deer in southern Texas for each latent period modeled as a uniform probability distribution. Results shown are from 100 simulations of the geographic automata model using the baseline deer surface.

	Area (km^2)					
Latent period	Median	% change, baseline	IQR	5%, 95%	Skewness	Kurtosis
1 – 5 days	4123	107.71%	576	1, 4755	-3.01	11.6
3 – 5 days	**1985**	**NA**	**139**	**1787, 2158**	**-0.499**	**4.45**
5 – 10 days	227	-88.56%	281	1, 326	-0.505	-1.77

Parameters and results in bold are from the baseline scenario

Table 6. Predicted number of deer infected from an outbreak of foot and mouth disease in a population of deer in southern Texas for each infectious period modeled as a uniform probability distribution. Results shown are from 100 simulations of the geographic automata model using the baseline deer surface.

	Number of deer					
Infectious period	Median	% change, baseline	IQR	5%, 95%	Skewness	Kurtosis
1 – 14 days	13063	-66.10%	16636	5, 20129	-0.358	-1.45
3 – 14 days	**38537**	**NA**	**2781**	**34785, 41829**	**-0.431**	**4.53**
14 – 28 days	36829	-4.43%	2542	32658, 40605	-0.191	3.578

Parameters and results in bold are from the baseline scenario

Table 7. Predicted number of locations infected from an outbreak of foot and mouth disease in a population of deer in southern Texas for each infectious period modeled as a uniform probability distribution. Results shown are from 100 simulations of the geographic automata model using the baseline deer surface.

	Area (km^2)					
Infectious period	Median	% change, baseline	IQR	5%, 95%	Skewness	Kurtosis
1 – 14 days	679	-65.79%	849	1, 1002	-0.395	-1.432
3 – 14 days	**1985**	**NA**	**139**	**1787, 2158**	**-0.499**	**4.45**
14 – 28 days	2114	6.50%	117	1903, 2345	0.276	3.523

Parameters and results in bold are from the baseline scenario

Table 8. Predicted number of deer infected from an outbreak of foot and mouth disease in a population of deer in southern Texas for each number of neighbors. Results shown are from 100 simulations of the geographic automata model using the baseline deer surface.

	Number of deer					
Neighbors	Median	% change, baseline	IQR	5%, 95%	Skewness	Kurtosis
4	3606	-90.64%	1447	16, 7063	-0.169	3.07
12	**38537**	**NA**	**2781**	**34785, 41829**	**-0.431**	**4.53**
28	119873	211.06%	3327	115106,	-0.126	3.07

Parameters and results in bold are from the baseline scenario

Table 9. Predicted number of locations infected from an outbreak of foot and mouth disease in a population of deer in southern Texas for each number of neighbors. Results shown are from 100 simulations of the geographic automata model using the baseline deer surface.

	Area (km^2)					
Neighbors	Median	% change, baseline	IQR	5%, 95%	Skewness	Kurtosis
4	205	-89.67%	77	1, 379	-0.329	3.07
12	**1985**	**NA**	**139**	**1787, 2158**	**-0.499**	**4.45**
28	6526	228.77%	249	6165, 6823	-0.393	3.23

Parameters and results in bold are from the baseline scenario

Table 10. Predicted number of deer infected from an outbreak of foot and mouth disease in a population of deer in southern Texas for each estimated population density surface. Results shown are from 100 simulations of the geographic automata model for each deer surface.

	Number of deer					
Population density	Median	% change, baseline	IQR	5%, 95%	Skewness	Kurtosis
Baseline	**38537**	**NA**	**2781**	**34785, 41829**	**-0.431**	**4.53**
Increase	48773	26.56%	3696	44118, 53278	-0.223	2.87
Decrease	29177	-24.29%	3091	25011, 33672	0.599	3.42

Parameters and results in bold are from the baseline scenario

Table 11. Predicted number of locations from an outbreak of foot and mouth disease in a population of deer in southern Texas for each estimated population density surface. Results shown are from 100 simulations of the geographic automata model for each deer surface.

	Number of deer					
Population density	Median	% change, baseline	IQR	5%, 95%	Skewness	Kurtosis
Baseline	**1985**	**NA**	**139**	**1787, 2158**	**-0.499**	**4.45**
Increase	2288	15.26%	163	2084, 2522	-0.103	2.79
Decrease	1650	-16.88%	166	1417, 1900	-0.923	3.72

Parameters and results in bold are from the baseline scenario

Table 12. Predicted number of deer infected from an outbreak of foot and mouth disease in a population of deer in southern Texas for each locally reduced (within a 10km neighborhood) estimated population density surface initiated from a high density index location. Results shown are from 100 simulations of the geographic automata model for each deer surface.

	Number of deer					
Population density	Median	% change, baseline	IQR	5%, 95%	Skewness	Kurtosis
Baseline	**56092**	**NA**	**2664**	**52248, 58606**	**-0.308**	**2.87**
-10%	52674	-6.09%	3086	48996, 57044	0.027	3.22
- 20%	50082	-10.71%	3662	38583, 54262	-4.067	19.242
- 30%	45926	-18.12%	6059	12, 52771	-2.756	9.814
- 40%	38901	-30.65%	8369	25245, 47225	-2.375	10.292
- 50%	27424	-51.11%	10727	9, 41833	-0.952	3.295

Table 13. Predicted number of locations infected from an outbreak of foot and mouth disease in a population of deer in southern Texas for each locally reduced (within a 10km neighborhood) estimated population density surface initiated from a high density index location. Results shown are from 100 simulations of the geographic automata model for each deer surface.

	Number of deer					
Population density	Median	% change, baseline	IQR	5%, 95%	Skewness	Kurtosis
Baseline	**2641**	**NA**	**132**	**2460, 2784**	**-0.308**	**2.77**
- 10%	2505	-5.15%	151	2325, 2714	-0.024	3.11
- 20%	2413	-8.63%	185	1867, 2611	-4.062	19.228
- 30%	2233	-15.45%	274	1, 2570	-2.793	9.979
- 40%	1934	-26.77%	382	1316, 2317	-2.606	11.453
- 50%	1435	-45.66%	500	1, 2091	-1.119	3.523

Table 14. Predicted number of deer infected from an outbreak of foot and mouth disease in a population of deer in southern Texas for each locally reduced (within a 10km neighborhood) estimated population density surface initiated from a low density index location. Results shown are from 100 simulations of the geographic automata model for each deer surface.

	Number of deer					
Population density	Median	% change, baseline	IQR	5%, 95%	Skewness	Kurtosis
Baseline	**6357**	**NA**	**8679**	**5, 11362**	**-0.069**	**1.619**
- 10%	4315	-32.12%	7042	5, 9697	0.241	1.663
- 20%	10	-99.84%	2850	4, 5626	1.20	2.88
- 30%	8	-99.87%	44	4, 5623	2.202	6.736
- 40%	3	-99.95%	5	3, 878	6.508	48.916
- 50%	3	-99.95%	7	3, 65	6.757	50.628

Table 15. Predicted number of locations infected from an outbreak of foot and mouth disease in a population of deer in southern Texas for each locally reduced (within a 10km neighborhood) estimated population density surface initiated from a low density index location. Results shown are from 100 simulations of the geographic automata model for each deer surface.

Population density	Number of deer					
	Median	% change, baseline	IQR	5%, 95%	Skewness	Kurtosis
Baseline	**590**	**NA**	**815**	**1, 1048**	**-1.068**	**1.581**
- 10%	413	-30.00%	661	1, 895	0.185	1.592
- 20%	2	-99.66%	294	1, 529	1.177	2.836
- 30%	2	-99.66%	9	1, 545	2.153	6.495
- 40%	1	-99.83%	1	1, 90	6.527	49.499
- 50%	1	-99.83%	2	1, 16	6.565	48.081

Table 16. Descriptive statistics for white tailed deer distributions predicted in south Texas, using information from the normalized difference vegetation index.

Variable	Observed	Mean	Std. Dev.	Minimum	Maximum	Skewness	Kurtosis
Baseline	30592	13	8	0	36	0.35	1.94
Winter	30592	13	6	1	28	0.61	2.28
Spring	30592	13	5	3	29	0.75	2.94
Summer	30592	13	4	5	27	0.54	2.92
Fall	30592	13	5	0	27	0.39	2.67

Table 17. Characteristics of spatial autoregressive lag models fitted to seasonal white tailed deer distributions in south Texas, derived using the normalized difference vegetation index.

Model	Parameters	Constant	NDVI	Rho
Winter				
	Coefficient	-1.41	6.06	0.918
	Std. error	0.028	0.096	0.003
	z-value	-14.67	21.08	284.7
	probability	<0.001	<0.001	<0.001
				Pseudo R^2 = 0.837
Spring				
	Coefficient	-1.2	5.2	0.932
	Std. error	0.105	0.305	0.003
	z-value	-11.36	17.04	313.3
	probability	<0.001	<0.001	<0.001
				Pseudo R^2 = 0.838
Summer				
	Coefficient	-0.88	4.17	0.938
	Std. error	-1.02	0.28	0.003
	z-value	-8.64	14.7	331.7
	probability	<0.001	<0.001	<0.001
				Pseudo R^2 = 0.838
Fall				
	Coefficient	-1.33	4.91	0.932
	Std. error	0.11	0.29	0.003
	z-value	-11.76	17.0	313.6
	probability	<0.001	<0.001	<0.001
				Pseudo R^2 = 0.838
n = 30,592; DF = 30,589				

Table 18. Predicted size of an outbreak of foot and mouth disease in a population of white tailed deer in southern Texas for each season by Ecoregion (Edwards Plateau [EP] and South Texas Brush [ST]). Results shown are from 100 simulations of the geographic automata model (Sirca) for each seasonal deer surface.

	Deer					Area (km^2)				
Season/Ecoregion	Median	IQR	5%, 95%	Skewness	Kurtosis	Median	IQR	5%, 95%	Skewness	Kurtosis
Winter/EP Plateau	101385	2868	98011, 104971	-0.19	-0.20	6416	154	6253, 6584	-1.9	9.3
Spring/EP	90913	2885	86699, 94147	-2.28	10.5	6050	139	5854, 6242	-3.3	16.3
Summer/EP	87792	2082	84955, 90341	-1.14	4.6	6058	131	5903, 6199	-3.4	22.5
Fall/EP	92323	2314	89212, 95410	-.92	2.07	6198	142	6019, 6360	-3.1	12.9
Winter/ST Plateau	40211	1819	36040, 41996	-2.9	13.9	4336	186	3890, 4541	-2.9	13.6
Spring/ST	50372	1330	43506, 52122	-2.9	10.1	4766	117	4009, 4946	-2.8	7.9
Summer/ST	54385	1753	52155, 56472	-4.7	29.8	4922	161	4752, 5122	-4.2	23.6
Fall/ST	53389	1546	51363, 54638	-3.01	11.7	4969	132	4786, 5077	-2.2	7.5

Table 19. Descriptive statistics for white tailed deer distributions predicted in an 8-county region of south Texas, and for distributions following application of 3 pre-emptive mitigation strategies.

Strategy	Regional estimated deer population	Deer, per km^2					
		Mean	SD	Minimum	Maximum	Skewness	Kurtosis
Baseline	406, 667	13	6	1	28	0.61	2.28
Targeted cull	289, 469	10	6	0	19	-0.25	2.45
Random cull	386, 202	13	6	0	28	0.29	2.62
Depopulation buffer	178, 373	6	8	0	28	1.03	2.85

Table 20. Predicted size of an outbreak of foot and mouth disease in a population of white tailed deer in an 8-county region of southern Texas for each of 4 mitigation strategies by Ecoregion (Edwards Plateau [EP] and South Texas Brush [ST]). Results shown are from 100 simulations of the geographic automata model (Sirca) for each mitigated deer surface.

	Deer					Area (km^2)				
Mitigation/Ecoregion	Median	IQR	5%, 95%	Skewness	Kurtosis	Median	IQR	5%, 95%	Skewness	Kurtosis
Baseline/EP	27000	1770	23927, 28781	-0.67	-0.19	1538	86	1385, 1623	-0.69	0.31
Baseline/ST	11863	829	10696, 12966	-0.51	0.98	1344	95	1211, 1472	-0.56	1.01
Targeted/EP Plateau	15406	484	14780, 15855	-9.07	84.8	969	27	933, 994	-9.2	86.2
Targeted/ST	11889	756	10687, 12665	-5.6	34.3	1345	87	1208, 1437	-5.6	33.6
Random/EP	24443	1290	22806, 25975	-0.69	1.17	1402	65	1324, 1476	-0.81	1.48
Random/ST	11293	727	10152, 12007	-6.9	59.2	1272	80	1146, 1361	-6.8	57.6
DB*/EP Plateau	12955	1088	11386, 14059	-4.6	22.3	795	55	713, 853	-4.8	23.7
DB*/ST	8226	360	7582, 8697	-5.1	25.1	938	44	860, 998	-4.9	24.7

* Depopulation buffer

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